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UNUSUAL CONDITIONS SIMULATING PULMONARY ATELECTASIS

WITH CASE REPORTS OF AGENESIS AND HYPOPLASIA OF THE LUNG AND OF FRACTURE OF THE BRONCHUS, TOGETHER WITH REPORT OF A CASE OF MASSIVE ATELECTASIS IN BRONCHIAL ASTHMA INCLUDED FOR COMPARISON

ROBERT E. PRIEST, M.D.

MINNEAPOLIS, MINN.

Unusual cases in which pulmonary atelectasis was the initial or "working" diagnosis are discussed here. Each proved to be some other condition on further study. Included in the case reports are: idiopathic pulmonary hypoplasia, pulmonary hypoplasia associated with congenital heart disease, pulmonary hypoplasia accompanying diaphragmatic hernia, unilateral pulmonary agenesis, and traumatic bronchial rupture. A case of total pulmonary atelectasis due to bronchial asthma is included for comparison. Bronchoscopy was of material assistance in the diagnosis in all of these cases, and bronchography using lipiodol was helpful in some.

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From the Division of Otolaryngology of the University of Minnesota Medical School, and the Ear, Nose and Throat Service of Minneapolis General Hospital.

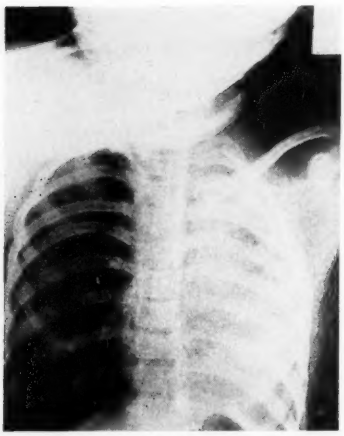

Fig. 3, Case 2.—(Idiopathic Pulmonary Agenesis). Bronchogram showing increased density of the left side of the chest with lipiodol filling the "clubbed" left main bronchial stump and outlining the trachea.

Fig. 4, Case 2.—(Idiopathic Pulmonary Agenesis). Lateral view showing lipiodol filling stump of left main bronchus and outlining trachea and some bronchi in right lung.

occurred during an asthmatic attack in an adult. Spontaneous pneumothorax, possibly due to rupture of the over-expanded non-atelectatic portion of this lung, accompanied the atelectasis. Massive atelectasis in an asthmatic child is described by Peshkin and Fineman[7] in 1931. These authors comment on the rarity of massive atelectasis in asthma in childhood in their experience. Seven cases of pulmonary atelectasis in asthmatic children are reported by Friedman and Molony.[8]

Atelectasis in asthmatic patients may be confined to part of the lung or may involve the entire lung. Cole, Nalls and Buis[3] describe several cases of partial atelectasis in asthma. Pulmonary fibrosis and bronchial stenosis are factors in producing atelectasis in asthma.[4]

Hilding[5] calls attention to the difficulty one has in understanding the mechanism by which atelectasis is produced when one reviews the various theories advanced to explain its existence. Coryllos and Birnbaum[6] conclude that atelectasis is caused by "bronchial obstruction with absorption of air distal to the obstruction". They speak of "bronchial plugs" of secretion as the usual cause of obstruction, and thus employ the term used by most modern writers. The formation of these "plugs" is clarified by a consideration of Hilding's[5] thesis. It is worth noting that Coryllos and Birnbaum[6] repeatedly emphasize that their use of the term "plug" should "not be taken to mean a cork-like occlusion at any one point in the bronchial tree, but refers to a variable column or mass of fluid, or semi-fluid, mucoid or fibrinous secretion or exudate.

Despite this adequate explanation, the bronchoscopist often is expected by the surgeon whose patient suffers from atelectasis, to retrieve a plug having definite form and shape which, in the mind of the average individual who thinks of atelectasis, has occluded the bronchus as a cork occludes a bottle.

In Case 1 there was no firmly fixed bronchial plug. In fact in no case of pulmonary atelectasis bronchoscoped by the author has a firmly fixed bronchial plug been found except in cases of acute laryngotracheobronchitis, and in foreign body cases. The customary finding has been a bronchus filled with thick, syrupy, gelatinous secretion resembling pus and sufficiently fluid to pass through an ordinary bronchoscopic aspirating tube. Usually the lung has aerated promptly after the fluid has been removed from the bronchial lumen.

By ingenious experiments with sections of trachea removed from freshly killed hens, Hilding[5] has demonstrated that diaphragms of

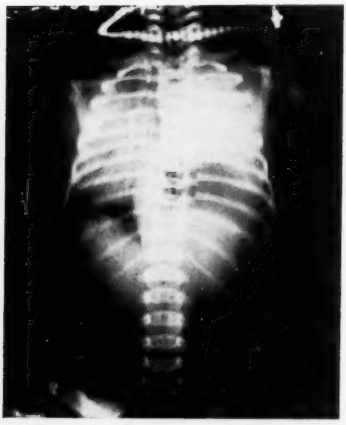

Fig. 5, Case 4.—(Diaphragmatic Hernia). Film shows what seemed to be atelectasis of the left lung and what actually was left sided diaphragmatic hernia. At Autopsy all abdominal viscera except liver, descending colon and rectum were found in the left thoracic cavity. Early thoracic surgery might have been life-saving; therefore early diagnosis is important.

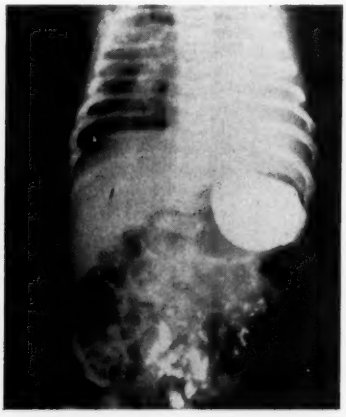

Fig. 6, Case 5.—(Pulmonary Hypoplasia Associated with Presumed Congenital Cardiac Defect). Film shows what seems to be atelectasis but bronchoscopy showed extremely small left main bronchus. Child died without permission for autopsy being granted so conclusive diagnosis could not be made.

mucus extending across the tracheal lumen are propelled by the cilia pushing air before them. The diaphragms move up the bronchi and trachea. Negative pressure is produced behind each diaphragm. In Hilding's experiment an equilibrium was reached at a point where the force exerted by the cilia was equalled by the sum of the air pressures within the bronchus. Hilding[5] believes that in a living lung the space having negative pressure would be filled in by over-expansion of adjacent normal portions of the lung, and that the elastic tissue of the affected portion of the lung would reduce its size. The increased flow of mucus brought about by the irritation of an anesthetic, or perhaps by reflex stimulus to secretory glands, would predispose to the formation of more mucus diaphragms which would pump out more air, and the process would continue until the affected lung or lobe or segment had been evacuated.

With this explanation in mind, one can understand the bronchus filled with thick mucoid material. This bronchus has become so full that the cilia can no longer move the load of mucus. Air distal to the mass undoubtedly is absorbed. In the bronchus occluded by extra-bronchial pressure or by a foreign body in the lumen, absorption of air is probably the major factor in production of atelectasis. But, as Hilding[5] says, "the piston-cylinder action just described very likely plays an important role also."

Case 2.—Idiopathic Pulmonary Agenesis. J. L., female, aged 4 years, normal and healthy, whose parents had noted enlargement of right thoracic cage and higher carriage of right shoulder than left. The family physician referred the patient to a pediatrician who ordered a chest radiograph expecting to find anomalies of the vertebrae to account for the thoracic asymmetry. Radiography showed what ostensibly was a total left pulmonary atelectasis, but in the absence of acute illness the radiologist considered the possibility of pulmonary agenesis. Bronchoscopy and bronchography confirmed this diagnosis (Fig. 3 and 4).

At bronchoscopy the trachea was found to be smaller in diameter than normal when an attempt was made to introduce a 5 mm bronchoscope. A normal trachea in a four year old child will admit a 5 mm scope quite readily. It was necessary to remove the 5 mm scope and use a 4 mm scope in this trachea. The trachea was normal, except for size, down to the bifurcation. It was obvious immediately that the bifurcation was abnormal. Instead of running in an antero-posterior direction, the bifurcation, ran from right to left as it proceeded anteriorly. The anterior end of the carina was therefore situated further to the left than was the posterior end.

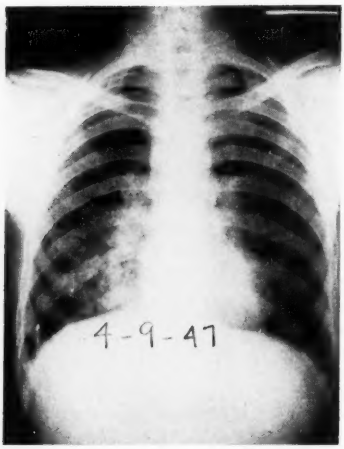

Fig. 7, Case 6.—April 9, 1947 (Rupture of Right Main Bronchus Due to External Chest Trauma). Radiograph of chest two months after severe chest trauma with crushing of thoracic cage and bilateral pneumothorax. The presence of old pleural adhesions prevented death by collapse of both lungs. Atelectasis is beginning in the medial portion of the right lung.

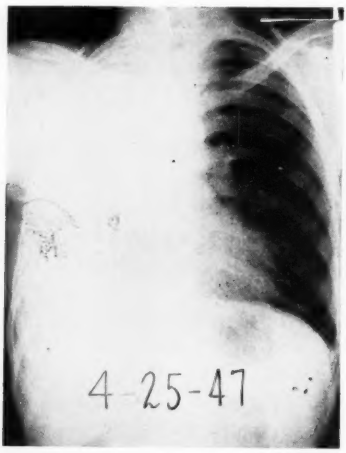

Fig. 8, Case 6.—April 25, 1947 (Rupture of Right Main Bronchus Due to External Chest Trauma). Radiograph showing what appears to be total atelectasis of right lung. Bronchoscopy on April 29, 1947 showed right main bronchus ending 1.5 cm below tracheal bifurcation. Forceps and bronchoscope were forced through the narrow point.

This abnormality was caused by the small size of the left main bronchus. A 3.5 mm scope (newborn type) was inserted instead of the 4.0 mm scope. The uppermost portion of the left main bronchus was seen to be about one-third the size of the right main bronchus. The left main bronchus appeared to end about 1.5 cm below the trachea. The end appeared to be a blind pouch. The right bronchial tree was inspected with the small scope and seemed to be normal throughout. Lipiodol was instilled and a bronchogram was made.

The patient was examined by a thoracic surgeon and no reconstructive surgery seemed possible. The child has continued to have a normal life despite her incomplete pulmonary equipment.

CASE 3.—Idiopathic Pulmonary Hypoplasia. T. McG., male, aged 6 years. The patient went to bed feeding well on May 7, 1948, and awakened whimpering. He complained of slight pain in his chest. The doctor in his home town x-rayed his chest and upon finding an increased density covering the entire left chest, attempted a pleural aspiration but obtained no fluid. The child was first seen by the author on May 8, 1948. He was not acutely ill but had dullness over the left chest, and x-rays again showed complete atelectasis of the left lung with shifting of the heart to the left and with a high diaphragm on the left. It was determined that bronchoscopy for diagnostic purposes and possibly for removal of a foreign body should be done.

Under intravenous sodium pentothal anesthesia administered by a physician-anesthetist, the trachea was entered with a 5 mm Jackson bronchoscope. At the bifurcation of the trachea an extremely small left main bronchus was encountered. A 4 mm scope was then introduced and an attempt was made to enter the left main bronchus with this instrument. This could not be accomplished because the bronchus was smaller than the instrument. A 3.5 mm newborn bronchoscope was then introduced and the tip of this instrument was small enough to enter the lumen of the left main bronchus at the bifurcation of the trachea.

The left main bronchus continued for a distance of approximately 2 cm below the bifurcation, growing smaller as it passed caudad until it ended in a dimple. The mucosa did not form a cup as if it closed a blind tube, but rather the mucosa of the entire circumference of the bronchus seemed sufficiently swollen to have obliterated the bronchial lumen. It seemed possible that there was a small bronchial lumen present normally which was closed by edema due to an acute inflammatory process.

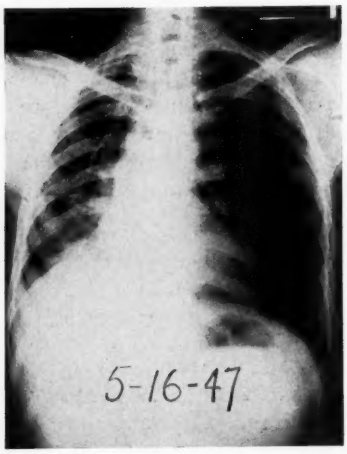

Fig. 9, Case 6.—May 16, 1947 (Rupture of Right Main Bronchus Due to External Chest Trauma). Radiograph showing re-expansion of right lung following bronchoscopy with forcible opening of right main bronchus.

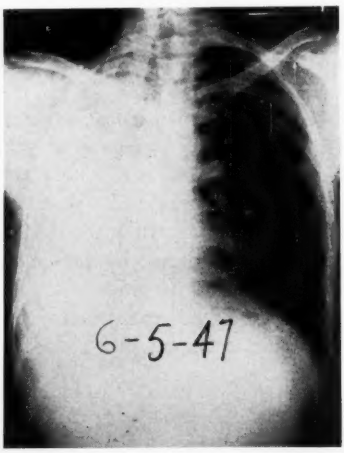

Fig. 10, Case 6.—June 5, 1947 (Rupture of Right Main Bronchus Due to External Chest Trauma). Radiograph showing almost total atelectasis of right lung again. On June 7, 1947 bronchoscopy demonstrated fractured ends of cartilaginous rings of right main bronchus about 1.5 cm below tracheal bifurcation. This stricture was again opened by forcing the bronchoscope through it.

The right lung was explored thoroughly with a larger bronchoscope and the right upper, the middle and the lower lobes, together with secondary divisions of the upper and the lower lobes were carefully inspected and found to be normal. One could see into the right upper lobe bronchus to the point where he clearly visualized two subdivisions. This is unusual and indicated a shift of the right upper lobe bronchus caudad. The thoracic surgeon could not recommend any reconstructive surgery, and the child has led a normal life since the illness just described, as far as is known.

Aplasia of the lung is rare. Recently excellent reviews of the subject have been published by Pierson[10] and by Mitchell.[11] The essence of the literature seems to be that about 100 proven cases have been reported; that the flaw producing the anomaly lies in the embryonic germ plasm; and that when unassociated with other anomalies incompatible with life, persons born with only one lung can survive to lead lives of normal length.

Cases of pulmonary aplasia have been reported in which there is no rudiment of a bronchus leading into the absent lung. Smart[9] in 1946 described two such cases in adult females. Foster-Carter[12] in 1946 distinguished between total aplasia in which there is no outpouching of any bronchus to the absent lung and cases in which there is a small outpocketing of the trachea representing the bronchus to the absent lung. He considers both as aplasia or agenesis. This is the condition in Case 2. Foster-Carter[12] classes cases having a rudimentary bronchus leading into a small fleshy structure without lobes as extreme hypoplasia. This is probably the condition in Case 3.

Classifications and nomenclature of pulmonary anomalies vary somewhat. Schneider[14] in 1912 made the following definitions:

a) Agenesis of a lung where there is complete absence of lung and bronchus.

b) Aplasia of a lung where there is a rudimentary bronchus and no pulmonary alveolar tissue. (Case 2 and 3 are examples.)

c) Hypoplasia of a lung or part of a lung where the alveolar tissue is underdeveloped.

The cases reported above are examples of total aplasia of the lung. In those following the lung was present but failed to develop to normal size because of other non-pulmonary anomalies which interfered with the growth of the lung.

It is of interest and of diagnostic importance to consider that accessory lungs can exist in chest and abdomen, not communicating

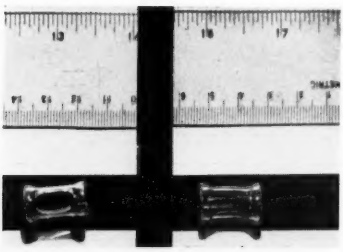

Fig. 11, Case 6.—(Rupture of Right Main Bronchus Due to External Chest Trauma). Photograph of plastic splint made to hold fractured bronchus open. Hole in splint was designed to allow air to enter the upper lobe bronchus. Splint could not be retained in position in absence of some means for producing fixation of splint.

Fig. 12, Case 6.—June 27, 1947 (Rupture of Right Main Bronchus Due to External Chest Trauma). Bronchogram showing lipiodol pocketing in the right main bronchial stump.

with the bronchial tree, and may be mistaken for tumors or dermoid cysts.[13]

CASE 4—Pulmonary Hypoplasia Associated with Diaphragmatic Hernia. Baby boy B., aged 7 hours, normal average birth. Child was born at 1:30 P. M. and was first examined by the author at 7:00 P. M. on the same day. The child had been cyanotic and had not breathed well from birth. An x-ray film suggested atelectasis of the left lung (Fig. 5, Case 4). The infant appeared to be in extremis.

Bronchoscopy was carried out immediately. The larynx was normal. A 3.5 mm Holinger infant bronchoscope was passed into the trachea. The left main bronchus was a narrow slit-like aperture and contained no secretion. The right main bronchus was normal and contained no secretion. The child died about one-half hour after the bronchoscopy was begun.

The autopsy report reads as follows.

CLINICAL DATA:

Baby lived for seven hours. Child breathed badly and showed intense cyanosis. X-ray of the chest was interpreted as showing evidence of only a small amount of air in the bases of both lungs. It was noted that there was no gas "in the stomach or intestinal loops." Bronchoscopy was carried out and a very small left bronchus was observed. Child died from respiratory distress.

AUTOPSY:

The body is that of a male infant. There is no external marking and no evidence of external malformation. There is no edema or jaundice.

The peritoneal cavity contains liver and the descending colon and the rectum. No other viscera are visible. The right pleural cavity is normal. The left pleural cavity contains all of the viscera except as noted above. The esophagus has entered the abdominal cavity and turns upward, so that the stomach is entirely inside the chest. The stomach is U-shaped with the duodenum near the esophagus and this loop is held in place by the common duct. The spleen lies on the left side of the pericardium. The left lung is posterior and in the apex of the cavity. The right pleural cavity shows no gross change.

The pericardial sac is negative. The heart shows no evidence of malformation, either in the vessels or the heart itself. There are fine, petechial hemorrhages on the visceral pericardia.

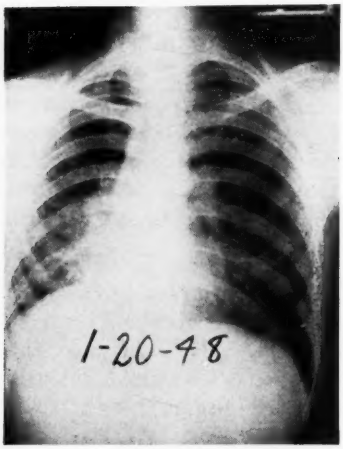

Fig. 13, Case 6.—Jan. 20, 1948 (Rupture of Right Main Bronchus Due to External Chest Trauma). Radiograph showing much greater aeration of right lung. Patient was clinically improved at this time, but improvement occurred without treatment and was difficult to understand in light of bronchial stenosis still present. (See next figure.)

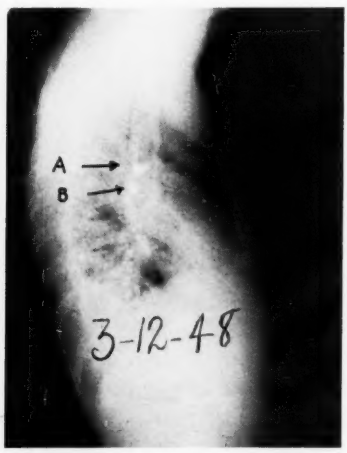

Fig. 14, Case 6.—March 12, 1948 (Rupture of Right Main Bronchus Due to External Chest Trauma). Bronchogram showing oil in bronchial stump "A" and in a tract "B" despite good clinical condition of patient and chest findings similar to those shown in Figure 13.

Lungs: The left lung and the bronchus are extremely small. The lung is leaf-like. There are two lobes. The bronchus and the pulmonary artery are both small. The right lung is atelectatic. There is air in the anterior and superior portions. There is no malformation.

The spleen weighs about 10 grams, is slightly large.

The liver is in its normal position and has normal lobulation.

The gall bladder is in its usual position. The common duct passes to the left and is elongated.

The position of the gastro-intestinal tract has been described above. When the bowel is replaced into the abdomen there is no apparent change. The viscera easily assume their normal positions and have a normal appearance except for the unusually long mesentery. There is no other malformation in the bowel. The pancreas lies inside the chest but otherwise is normal. Right adrenal and kidney show no gross change. The left kidney and adrenal show a normal form but they are displaced into the left pleural cavity. They lie in what corresponds to the retroperitoneal tissues and are not pedunculated into the pleural space.

Ureters and bladder show no malformation.

Diaphragm: The right half of the diaphragm is intact. The esophagus passes through its hiatus in a normal fashion. The defect in the left half of the diaphragm is posterior and lateral and only a narrow, crescentric, sickle-shaped strand of muscle is present anteriorly and laterally.

Head: Not examined.

Conclusion: Malformation of diaphragm.

This case is reported because early diagnosis of diaphragmatic hernia makes possible the saving of some of these babies by prompt surgical correction of the anomalous position of the abdominal viscera, and repair of the diaphragmatic defect.

An exactly similar case is reported by Foster-Carter.[12] In his case "the left half of the diaphragm was absent, and there was a pleural defect in the anterior mediastinal region; the left pleural cavity contained the stomach and most of the intestines; a loop of small intestine extended into the right pleural cavity through the anterior mediastinal defect. The right lung was expanded and appeared normal. The left main bronchus was very small and led into a tiny lung about the size of a hazel nut, which had a shallow fissure. Bronchography revealed

a branching bronchial system in the hypoplastic left lung; the right bronchial tree was normal".

Excellent discussions of diaphragmatic hernia are given by the Rubins,[15] Liebow and Miller,[16] Harrington,[17] and Bremer.[18]

Case 5—Pulmonary Hypoplasia Associated with Congenital Cardiac Defect. J. C., male, aged 2 months, had been cyanotic since shortly after birth. Cardiac murmurs suggested the presence of congenital cardiac defects. A bronchoscopy was decided upon to rule out pulmonary abnormalities when chest x-ray (Fig. 6) showed what seemed to be total atelectasis of the left lung.

The bronchoscopic findings were: The larynx was exposed with an infant Flagg laryngoscope and was normal. A 3.5 mm bronchoscope was introduced into the trachea. The trachea was normal down to the bifurcation.

The right main bronchus was normal and the scope was not introduced into it except at its very upper end.

The left main bronchus was represented by a dimple in the tracheal wall. Leading downward from the dimple was a tiny tube which breathed like a normal bronchus. The diameter of this tube was not more than 0.75 to 1.0 mm.

Diagnosis: Marked constriction, left main bronchus, not due to external compression unless such compression has been present since before birth; probably is more properly called aplasia of left main bronchus but is not a true agenesis because there is a lumen and it does seem to conduct air. Bronchogram was not done for fear of causing respiratory embarrassment.

The child left the hospital and died shortly. Autopsy permission was refused.

Case 6—Rupture of Right Main Bronchus Due to External Chest Trauma (Fig. 7 through 14).

A. W., white male, aged 34 years, was crushed in a "clam-shell" type of mechanical shovel used in excavating debris from a burned grain elevator early in 1947. The patient suffered bilateral pneumothorax, but old pleural adhesions prevented total collapse of his lungs. He had marked subcutaneous emphysema and was in a severe state of shock. He responded well to supportive therapy and was released from the hospital several weeks after the accident. The patient was quite well for a few weeks when he noted malaise, dyspnea and fever. Chest x-ray showed what was apparently a total atelectasis of the right lung.

Bronchoscopy on April 29, 1947 showed a normal trachea and tracheal bifurcation. The right main bronchus was filled up to the carina with grey viscid secretion. This was removed with suction and the bronchial lumen was found to terminate 1 or 1.5 cm below the tracheal bifurcation. The lumen narrowed down, and an area to the right and posterior looked "thin." A bronchoscopic forceps was pressed gently against this area and in a moment went through into the lower portion of the right main bronchus. After the bleeding following this maneuver had subsided it was decided to advance the bronchoscope through this lumen. The patient seemed to breathe through this new lumen which was then about 2 mm. in transverse diameter and about 1 mm in antero-posterior diameter. The bronchoscope beak was inserted into the new lumen and the scope was cautiously advanced until it suddenly jumped forward into the bronchus below the point of stricture. There was again some bleeding but this soon subsided. The bronchial mucosa was thick and had a deep wine color. There was no large amount of secretion down below the stricture. After the bleeding had stopped it seemed that the patient breathed beyond the former point of stricture and down into the lower portion of the lung. The patient left the operating room in good condition. A diagnosis of occlusion of the right main bronchus of undetermined cause, possibly rupture of the bronchus, was made.

After the bronchoscopy done on April 29, 1947 the right lung re-expanded until most of the lung had re-aerated. The air-containing portions of the right lung began to collapse in 10 days or 2 weeks and on May 28, 1947 it was necessary to bronchoscope the patient again. The findings were identical with the original bronchoscopy, i.e., the bronchial stricture had re-formed and air could no longer enter the right lung. The patient was bronchoscoped on June 7, June 14 and June 30, 1947. The narrowing of the bronchus would recur between the bronchoscopies and would be temporarily opened by each bronchoscopy.

On June 14, 1947 an attempt was made to insert a plastic spool-like splint (Fig. 11) to hold open the crushed portion of the right main bronchus.

This splint was not retained in position and it is rather doubtful that it could have accomplished its intended purpose even if it had stayed in position.

Lipiodol studies of the lung were made on June 27, 1947 and showed a complete occlusion of the right main bronchus. Lipiodol studies were made following the bronchoscopy on June 30 and oil

ran down into the right main bronchus (the lower portion) but did not fill the sub-branchings within the middle and the lower lobes. The right upper lobe had had some air in it from the time of the first bronchoscopy and had never totally collapsed after the first bronchoscopy. The route of entry of this air was never demonstrated.

Rupture of a bronchus is uncommon as a complication of external chest trauma. The subject has been completely reviewed recently by Kinsella and Johnsrud.[19] Thirty-eight cases had been reported prior to the report of Kinsella and Johnsrud.[19] They reported two cases and Holinger et al[20] added three more cases. The case reported here (Case 6) was also encountered in Dr. Kinsella's[19] practice. It seems likely that this condition may have been overlooked at times and that it may be found more often if the possibility of its existence is considered when there is severe chest trauma.

As far as is known the insertion of an endobronchial splint as attempted in this case is here reported for the first time. Retention of such a splint is a very puzzling problem, and it would seem that suturing such a splint into place would offer some promise. This has been done experimentally in the trachea by Longmire.[21]

The treatment of bronchial narrowing resulting from bronchial rupture has consisted either of observation or of repeated dilatation. The case reported here is typical in its story of repeated bronchoscopy with final failure to maintain a patent lumen. It seems that either resection of the ruptured bronchial rings and end-to-end suture of the intact portions of bronchus on either side of the rupture, or insertion and retention of an endobronchial splint, offer the only real chances for cure in cases of bronchial fracture.

302 Medical Arts Building.

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LXXXI

THE ENDOSCOPIST AND THE NEWBORN INFANT

IRL H. BLAISDELL, M.D.

SYRACUSE, N. Y.

The neonatal period is considered, for the purposes of this paper, as the time interval from birth until the fourteenth day of life. This period is always a critical one, peculiar, in that it is the result of physiological adjustments accompanying birth, or that it is due to the accidents of labor. Many congenital malformations and anomalies are seen only during the neonatal period, either because they tend to be outgrown, or because their serious nature is incompatible with prolonged life.

When a newborn infant fails to respond to the usual methods of resuscitation and congenital anomalies are suspected, the situation becomes a grave emergency which demands all the technical skill and diagnostic acumen of the obstetrician and the pediatrician. It is at this point that the endoscopist may be called in consultation.

The endoscopist is well trained to handle some of these problems, particularly when they are of a surgical nature. However, he should be of further assistance to his colleagues by helping to make the diagnosis, deciding whether or not surgical procedures are indicated, and by rendering his opinion as to prognosis and ultimate therapy later in life. The diminutive size of the patient in such cases should present little technical difficulty to the trained endoscopist, but certain fundamental rules familiar to all pediatricians must be borne in mind; these will be discussed later.

Unfortunately, several factors prevent the endoscopist from being called as frequently as he might be. First of these is the fact that many obstetricians and pediatricians do not realize the help which the endoscopist can often give them. They, therefore, are content to continue in their time-honored methods, sometimes even failing to make a diagnosis at all.

Second, a number of obstretricians and pediatricians have had the experience of calling an endoscopist in consultation, only to find

that they received little or no contribution to the problem at hand, or that poor judgement on the part of the consultant added further complications to an already difficult clinical situation. Thus, the pediatric and obstetrical staff come to feel that such endoscopic consultations are quite unnecessary, and that they can do a better job by themselves.

The third factor concerns the attitude of the endoscopists. Very often the crises which occur in the newborn infant are real emergencies. To be of any value, immediate availability of the endoscopic consultant is of primary importance. The obstetrician not infrequently finds himself confronted by a serious problem in the early hours before dawn, at a time when the endoscopist is reluctant to leave the warmth and comfort of his bed. If he fails to respond to the call of distress of his colleagues, preferring to render his opinion during his regular hours for hospital rounds, the obstetrician in all future cases will train his own staff to carry out whatever measures may be necessary with the equipment available. Thus, the endoscopist, by his being unavailable, will seldom see the group of pathological problems which can arise during the neonatal period.

The conditions in the newborn which are of interest to the endoscopist are, of course, those dealing with the air and food passages. A few of these diseases call for operative procedures, and yet the opinion of the endoscopist can be of considerable value even in the non-surgical conditions. In this regard, a general knowledge of other pathological entities, sometimes only remotely related to the tracheobronchial tree, is a definite asset. To this end a brief review of some of the causes of respiratory difficulties in the newborn will be discussed with their points of differential diagnosis.

It has been shown that respiration during fetal life is carried on by the placental circulation, and rhythmic but feeble respiratory movements are known to occur in utero. It is believed that such movements occur with a closed glottis. More marked ones have been noted during parturition, when the fetal circulation has been occluded; but as a rule, the first deep inspiration is made only after birth.

At birth, the normal infant begins to cry and use his arms and legs, and the purplish color of the skin gives way to a rosy pink. In the first stage of asphyxia, however, the newborn child is deeply cyanosed and there is no attempt at respiration, or only superficial

efforts repeated at long intervals. In the second degree of asphyxia, the picture is that of surgical shock. The child is pale, the heart action weak, irregular and slow, and the extremities are limp and cold. The heart action becomes more and more feeble until it ceases.

In the literature, the term "asphyxia neonatorum" has been used to include a wide variety of conditions which lead to deficient oxygenation of the blood. Primary respiratory failure, intracranial injuries, congenital cardiac and vascular anomalies, and lesions producing obstruction to respiration are all possible causes of neonatal asphyxia. Some knoweldge of these entities as to symptomatology, therapy, and prognosis, must be possessed by the endoscopist if he is to satisfactorily handle his role of consultant.

PRIMARY RESPIRATORY FAILURE

Failure of the newborn infant to breathe properly after delivery may occur as a result of faulty development of the respiratory center itself. This is thought to be the case with the smallest premature infants, but as a rule it is considered to be the result of injury at the time of parturition. In addition, there are certain factors which interfere with the placental circulation and thus produce cerebral anemia and failure of the respiratory center to function. Some of the conditions which produce this interference with circulation are a prolonged second stage of labor, the use of pituitrin preventing relaxation of the uterus in the second stage, maternal convulsions or hemorrhage, early separation of the placenta, winding of the cord about the child's neck, and, finally, the death of the mother. Rarely, the respiratory system may be stimulated before birth, due to the increased venosity of the blood, and aspiration of the amniotic contents then occurs. This does not interfere with respiratory movements but does produce obstructive symptoms in those infants who survive.

Organic lesions of the nervous system, particularly cerebral hemorrhage, and occasionally damage to the phrenic nerve resulting from birth injury, are sometimes responsible for respiratory failure in the newborn.

It is often difficult to determine the cause of respiratory failure. A reliable obstetrical history is, of course, important. The chief problem is to decide whether or not an organic lesion is present. It is often impossible to decide this question at once, since the signs

of intracranial hemorrhage, for example, may not appear until 36 to 48 hours later.

Frequently enough, the respiratory center is depressed by anesthetics and analgesics administered during labor. If the poor respiratory efforts are due to the use of such agents, the administration of oxygen is imperative. The general consensus of opinion is that carbon dioxide should not be used in conjunction with oxygen in these cases. Above all, the infant should be kept warm during the resuscitation period and thereafter. Stimulating drugs are generally frowned upon as doing more harm than good, but many pediatricians advise their use to overcome the effects of morphine and the barbiturates in very sleepy babies.

The establishment and maintenance of an airway is the first requirement in resuscitation of the newborn, no matter what the cause of the asphyxia may be.

INTRACRANIAL BIRTH INJURIES

Cerebral edema from trauma during labor accounts for at least part of the picture of shock so often seen in the newborn. Evidence of intracranial hemorrhage is found in approximately 30% of infants who are still-born or die within two weeks.[24] Intracranial hemorrhage may occur in spontaneous easy deliveries, and even occurs in infants born by cesarean section. Most often, however, such trauma arises from the complications of labor. Post-term infants, because of their size, are particularly subject to injury. The symptoms: If hemorrhage is large, the child is usually still-born; if the degree of bleeding is not severe enough to be immediately fatal, the infant may be born deeply asphyxiated and respiration established only after considerable difficulty.

The symptoms of birth injury may be present immediately after birth, or on the other hand, may occur only after an interval of several days due to late bleeding. There is usually cervical rigidity, cyanosis, a feeble cry, failure to nurse and continuing loss of weight. Intermittent cyanosis is characteristic. Convulsions are common, and respiration is slow and irregular. The diagnosis is obvious when the typical symptoms of convulsions, rigidity and cyanosis are present but in milder cases is more difficult. Irregular respirations, the inability to nurse, and intermittent cyanosis are suggestive signs of intracranial injury. Atelectasis may cause intermittent cyanosis but,

as will be noted later, atelectasis is, in itself, the result of imperfect respiratory function caused by hemorrhage. An examination of the spinal fluid by the pediatrician should be of considerable help in making the diagnosis.

CARDIAC AND VASCULAR ANOMALIES

Some of these malformations are of such a severe character that they are incompatible with life. The diagnosis of congenital heart disease is made more difficult by the fact that the absence of a heart murmur does not preclude the existence of an anomaly, since many circulatory disturbances give no murmurs, while accidental murmurs having little significance may frequently be found. In general, the cyanosis produced by cardiac disease does not yield readily to oxygen by inhalation. The opinion of the pediatrician in these cases can give the endoscopist a great deal of comfort when the cyanosis can be satisfactorily explained on a cardiac basis.

RESPIRATORY OBSTRUCTION IN THE NEWBORN

There are a number of conditions which produce obstruction to respiration, some of which are only remotely related to the tracheobronchial tree. Among the malformations of the nose and throat which may lead to respiratory obstruction is atresia of the posterior choana. This may not be detected at birth and may only be noted a few days later when the child finds difficulty in nursing and is overwhelmed by an abundance of secretions which tend to interfere with respiration. The diagnosis can be made by passing a soft rubber catheter through the nose and finding an obstruction, or by the instillation of a small amount of iodized oil in the nose, followed by x-ray studies which reveal the cause of the obstruction. In the same way, dermoid cysts and ranula of the floor of the mouth may cause obstruction to breathing and swallowing. These anomalies are rare.

Congenital malformations of the larynx are somewhat more common, and give rise to a clinical picture which is quite characteristic and passes under the name of congenital laryngeal stridor. The symptoms are seldom present at birth but usually begin during the first few days of life. There is an inspiratory stridor which may be very pronounced, accompanied by retraction of the soft parts of the chest wall. There is usually no cyanosis or subjective distress and the child appears fairly comfortable. Expiration is easy and the

voice and cry are generally normal. The stridor becomes intense with excitement and crying, and at this time there may be cyanosis and dyspnea. The stridor diminishes when the child is quiet but usually does not completely disappear even in sleep. Diagnosis can only be made by direct laryngoscopy and the laryngeal findings vary widely. Frequently there is an exaggeration of the normal infantile characteristics, associated with flabby aryepiglottic folds, which tend to collapse and be drawn into the glottic chink with inspiration.

The epiglottis, in some instances, is unduly long, while in others it may be broad and flaccid. An abnormally small larynx has also been described. Paralysis of the vocal cords as a result of birth trauma has also been reported. The obstruction may be found subglottic due to malformations of the trachea, either with absence of the tracheal rings or other abnormality of the cartilages of the trachea, or redundant mucosa. Congenital cysts of the larynx, however, usually do not produce symptoms until later in life. However, they may cause death if the larynx becomes obstructed by the mass present at the base of the epiglottis.[16]

The symptoms of congenital cysts of the larynx, as pointed out by Holinger,[3] depend upon their position and size. They may produce stridor and retractions of the chest wall with occasional cyanosis. The latter is not improved by the administration of oxygen. Congenital cysts are typically found in the lateral wall of the larynx or in the aryepiglottic fold, and must be distinguished from prolapse of the ventricle.

Congenital stenosis of the larynx has been reported by Jackson.[25] In these cases the infants were still-born. Congenital laryngeal webs also occasionally occur and give rise to the symptoms of laryngeal stridor. The child has dyspnea and a croupy and aphonic cry. Cough also is often present. The diagnosis of congenital malformations of the larynx can of course only be made by direct laryngoscopy.

However, there is an interesting group of cases which may simulate obstructive dyspnea of laryngeal origin. Symptoms similar to laryngeal obstruction may occasionally be produced by enlargement of the thymus and such cases have been found to disappear following irradiation. Infants with a recessive chin and hypoplasia of the mandible have stridor which is not produced within the larynx, but is the

result of the tongue falling backwards and interfering with the entrance of air into the larynx.

In those cases in which laryngeal obstruction is caused by some temporary factor related to immaturity of the tissues or the neuromuscular mechanism, the obstruction is relatively benign and often self-limited. After a few weeks the stridor becomes stationary and generally after the first six months of life tends to diminish. Although it may be impossible at first to distinguish abnormalities of a more permanent nature from those which are benign, the factor of time, and the failure of the symptoms to diminish, points to a permanent type of defect. This is true of congenital webs and subglottic obstructions caused by stenosis of the trachea, or papillomas, or, rarely, a mediastinal tumor. The prognosis in most of these congenital cases is good, the chief danger being from intercurrent infection, such as pneumonia.

PERSISTENT ATELECTASIS

Under normal conditions, the expansion of the lungs takes place readily and is usually complete in two or three days. The sequence of events is as follows: The anterior portions of both lungs are the first to be inflated, followed by the upper lobes and finally the lower lobes posteriorly. The superficial nodules are nearly always expanded before those in the interior of the lungs. Delayed expansion is generally the result of early respiratory failure. Atelectasis may be found in cases in which respiration has been initiated spontaneously, not because of prematurity of the infant, but because inspiration has continued with insufficient force and depth to produce full expansion. Malformations of the thorax or diaphragm, or abdominal distention by fluid or gas, may impede pulmonary expansion. The process is nearly always symmetrical. The unexpanded portions being those which are normally the last to expand, that is, the posterior portions of the lower lobes.

Atelectasis may give no symptoms whatever, and a newborn infant may live for two to three weeks without cyanosis or dyspnea, although using only one-third of his pulmonary capacity. The characteristic finding is cyanosis of a periodic rather than a continuous type. The physical signs are not prominent. The process is bilateral. There is usually good resonance of the entire chest due to emphysema of the expanded areas surrounding the collapsed areas.

When atelectasis of one side occurs, dullness and diminished expansion may be transmitted with abnormal intensity. X-ray studies may reveal irregular shadows at the base, but nothing very characteristic. However, if atelectasis is unilateral, the picture is more striking and generally shows consolidation of the involved side with marked compensatory emphysema of the other, usually with a shift of the mediastinum. Atelectasis should be suspected in infants where difficult resuscitation is followed by feeble and irregular attempts at breathing. This is especially true of premature infants. Cyanosis which occurs in attacks and is relieved when the child cries vigorously is generally atelectatic in origin.

Congenital atelectasis may be confused with pneumonia which often co-exists, and must also be differentiated from cyanotic attacks due to congenital heart disease. The cyanosis of atelectasis yields more readily to inhalation of oxygen than do cardiac anomalies associated with a venous shunt.

BRONCHIAL OBSTRUCTION

Bronchial obstruction may occur at any time after birth from the aspiration of foreign material, either a feeding or vomitus. Sudden violent choking attacks are produced as result of aspiration. If the patient survives immediate asphyxia, he is prone to develop aspiration pneumonia.

In contrast with aspiration of relatively small amounts of foreign material, there is a dramatic picture produced by massive aspiration of amniotic contents. A minimal amount of such aspiration occurs normally and amniotic fluid is generally present in the mouth and pharynx and probably to some degree in the trachea and large bronchi as well. In order for massive aspiration of amniotic contents to occur, regular deep respirations must have developed before delivery. This condition was thought to be rare but was found by Farber and Wilson to occur in 15% of deaths of full-term infants up to two days of age. The causes of respirations in utero are, of course, those of fetal asphyxia in general; a dry labor should be mentioned, in particular, since this not only favors compression of the cord, but gives additional cutaneous stimulation of the fetus.

Brochial obstruction may not be obvious at birth because of the fact that the aspirated material at first lies loosely in the tracheobronchial tree and, therefore, does not afford as serious an ob-

stacle to respiration as it does later when it becomes dry and impacted, and forms a thin membrane lining the tracheobronchial tree. Such a membrane consists primarily of vernix, in which are imbedded cornified epithelium from the skin of the fetus, and polygonal amniotic cells. As the vernix membrane becomes impacted evidence of bronchial obstruction becomes conspicuous. In these cases, the prognosis is always a doubtful one, as many of the children succumb during the early days of life. The course of events is one of increasing cyanosis while the respiration becomes rapid, with inspiratory retraction of the soft parts of the chest. The contrast between the violence of the respiratory movements and the small amount of air entering the lungs is striking. This produces a marked exaggeration of the negative pressure in the thorax during inspiration and as a result, rupture of the expanded alveoli may be produced. A complication much to be feared is pneumothorax.

PNEUMOTHORAX

Pneumothorax in the newborn is most frequently produced by incomplete bronchial obstruction from aspirated amniotic contents. However, plugs of mucous, excessive pressure used in artificial respiration, or injuries to the chest wall from obstetrical procedures and instruments may also give rise to the condition. Pneumothorax should be suspected when the infant develops sudden dyspnea and cyanosis, often accompanied by restlessness and shock. Physical signs are generally of little help in making the diagnosis. Occasionally, a shift of the mediastinum and hyper-resonance of the affected side may be found. Pneumothorax in most readily confused with diaphragmatic hernia and lung cysts, but the latter can be distinguished by x-ray studies.

CONGENITAL CYSTS OF THE LUNG

Congenital cysts may be either single or multiple. Occasionally they communicate with the bronchi. The areas surrounding these cysts are often atelectatic. If lung changes are extensive, symptoms of asphyxia are present and such infants do not survive but a week or two. If the cysts are small and the atelectatic process is not extensive, there may be no symptoms for a time. However, these patients generally succumb to intercurrent infections in early life.

The diagnosis of pulmonary air cyst is not difficult if the possibility of such a condtion is suspected. As pointed out by Anspach

and Wolman,[22] the ease of confusing diaphragmatic hernia or eventration of the diaphragm with pulmonary air cysts can be easily overcome by the ingestion of small amounts of barium, and demonstrating the abdominal viscera either in their normal, or abnormal positions. Single large cysts may also be confused with pneumothorax but the lower position of the hilus and the fibrillar structures inside the cysts by x-ray, help to differentiate them.

Because of the urgent symptoms and extremely poor physical condition of these infants, the importance of speedy action rather than prolonged study becomes obvious. In some cases, the infant progresses normally for a few days and then becomes increasingly dyspneic and cyanotic. This is aparently due to the fact that the bronchial communication with the cystic areas is oblique or distorted, forming a valve-like opening which traps increasing amounts of air, and balloons out the cavity more and more, at the same time displacing the effective lung parenchyma until pulmonary decompensation occurs. Further evidence for this is found in the positive pressure within the cyst, the temporary improvement from aspiration or drainage, and the disproportion between the size on x-ray views and that of the removed specimen. Burnett and Caswell[7] have reported lobectomy for pulmonary cysts in a fifteen day old infant with recovery.

MALFORMATIONS OF THE ESOPHAGUS

Congenital abnormalities of the esophagus may give rise to symptoms of disturbed function of both the respiratory and gastrointestinal tracts. It is a very frequent occurrence for normal babies to regurgitate some of their food, and ordinarily it is of little consequence. However, when the newborn infant, shortly after delivery, starts to bring up mucous and chokes, and becomes cyanotic when trying to swallow, it is a matter of grave concern to the attending physician, and the possibility of atresia of the esophagus should be considered. The most common congenital anomaly of the esophagus is complete atresia, in which the upper segment communicates with the trachea near the bifurcation. Eighty-six percent of the esophageal anomalies fall into this group.[9] There may be suffocating attacks with cough and cyanosis and even a fatal outcome. Drooling is often conspicuous, and gastric tympany is present as a result of the bronchoesophageal fistula. Esophageal fistula without

atresia is a very rare form of malformation producing periodic choking attacks without difficulty in swallowing.

The diagnosis of atresia of the esophagus can be made by passing a small rubber catheter into the esophagus, until it reaches the obstruction in the upper third, or fails to enter the stomach. However, this may give misleading information unless carried out under the fluoroscope, as the catheter may double back on itself in the dilated upper esophageal pouch so readily as to give the impression it is passing through the esophagus into the stomach.[11] Iodized oil may also be instilled into the esophagus to demonstrate the upper segment. The oil, however, should be aspirated after the x-rays have been taken, in order to prevent aspiration into the tracheobronchial tree. If too great a quantity of the opaque solution is placed in the oral segment, it may be aspirated into the lower portion of the trachea, and subsequent x-rays show the material below the level of the blind pouch, thus suggesting a stricture of the esophagus rather than a true atresia.

Bronchoscopy has been recommended by Shaw[23] as a diagnostic aid, although it has not been uniformly accepted as essential. However, it is important to locate the exact site of the fistula. It has been pointed out, also, by most writers that neither a low position of the fistula, nor a high level of the upper segment contraindicates surgical investigation since exploratory thoracotomy is the only efficient method of determining the possibility of direct anastomosis of the esophageal segments.[12] Such conditions as malformations of the esophagus are extremely serious and demand sound judgment and prompt action, for if left alone, death from inanition will follow within a very few days, associated frequently with a terminal pneumonia.

The treatment for congenital esophageal atresia with tracheoesophageal fistula is surgical and more and more cases are being reported by thoracic surgeons who have successfully repaired the defects, either by transpleural esophageal anastomosis or esophagogastrostomy.

Webs of the esophagus can frequently be treated by breaking the web with the esophagoscope, and esophageal stenosis and cardiospasm severe enough to warrant treatment will respond to esopha-

geal dilatation. Cardiospasm occasionally occurs in children but rarely in infants.

TREATMENT

Usually by the time the endoscopist is called in consultation for a newborn infant who is having respiratory difficulty, several hours or days may have elapsed since birth. Generally the obstetrician or pediatrician has followed out such routine measures as were indicated for resuscitation. These, of course, include the aspiration and removal of secretions from the pharynx, the administration of heat, either in the form of a warm blanket or a warm bath; gentle artificial respiration may have been instituted in addition to the administration of oxygen, either with or without carbon dioxide. Other methods of resuscitation, as spanking, cold baths, vigorous artificial respiration and Saultze's method of swinging, only tend to increase the degree of shock or the degree of intracranial injury and are mentioned only to be condemned.

The first problem which confronts the endoscopic consultant is one of accurate diagnosis as to the cause of the respiratory failure. Again, the importance of a reliable obstetricial history should be stressed. X-ray studies of the chest are very desirable and helpful if time and the general welfare of the patient permit. The endoscopist should first ascertain whether or not the pathology is in the mouth, pharynx, or upper respiratory tract. If the structures in these regions are normal, intracranial damage and cardiac and vascular anomalies should then be ruled out. It is here that the pediatrician can be of the greatest assistance.

The endoscopist will now have to decide whether or not some endoscopic procedure is indicated and warranted. An accurate diagnosis of laryngeal pathology cannot be made without direct laryngoscopy. Such a procedure, properly carried out, should yield much in the way of helpful information, without trauma to the infantile structures. If the consultant decides that aspiration of the tracheobronchial tree is indicated, this may be carried out by the use of the newborn aspirator with a flexible rubber tip. Such an instrument can be passed easily through the glottis with a minimum of trauma, and often enough will give gratifying results by the aspiration of secretions which otherwise could not well be removed. Obviously, such a procedure does not give the operator an opportunity

to inspect the structures of the tracheobronchial tree. When such inspection appears necessary, the infant bronchoscope of 3 to 3.5 millimeters in diameter may be used. Such manipulation, needless to say, must be done with the utmost gentleness and the procedure must not be unnecessarily prolonged if trauma to the larynx is to be avoided.

Because of the gravity of the problem of resuscitating newborn infants, obstetricians have become increasingly aware of the necessity for aspirating the tracheobronchial tree without undue delay. Stringer[14] advises the use of a laryngoscope of the Flagg type by a member of the anesthesia or obstetrical department, and aspiration with a soft rubber catheter held by a special forceps which does not completely close the catheter. He states, "In adding this resuscitation measure to our obstetrical service we feel that results have justified its adoption as a routine procedure."

Sullivan, writing on anoxia neonatorum, stresses intubation with suction, followed by insufflation with oxygen. He states, "Intubation may be indirect with a catheter, or direct by the use of the laryngoscope, depending upon the experience of the operator and the equipment available. The latter is preferable."[18]

Everyone is agreed that the tracheobronchial tree should be aspirated when there is evidence of atelectasis or bronchial obstruction. Bronchoscopy should be carried out when there is evidence of pneumothorax and massive bronchial obstruction, and may also be of considerable value in some of the more uncommon pathological entities that have been mentioned.

Routine bronchoscopy and aspiration of all cyanotic infants is unnecessary and may lead to further difficulties. Each case should be decided on its own merits after all factors producing the cyanosis have been evaluated. However, routine aspiration of the pharynx and tracheobronchial tree by means of a flexible tipped suction, or a catheter, in all infants who shows signs of respiratory failure and asphyxia, is worthwhile. "Of 95 cases delivered (by section) in a New Orleans hospital, without intratracheal catherization, nine died. Of 102 deliveries followed by routine catherization, two died. Routine intratracheal catherization may, therefore, be a life-saving measure for an infant delivered by cesarean section."[17]

CONCLUSIONS

The mortality rate in newborn infants is high, and the chief cause of death is asphyxia, as shown by the statistics of the Boston Lying-In Hospital, where deaths from this cause accounted for 58% of the total infants autopsied since 1931.

The danger of prolonged asphyxia is shown by the figures of Schreiber who has collected well over 1,000 cases of children with spastic diplegia, cerebral atrophy, epilepsy, mental deficiency, and other nervous lesions in which the only apparent causative factor was severe asphyxia at birth.

The assistance which the endoscopist can give the obstetricial and pediatric services in the handling of problems of resuscitation and aspiration of the newborn is generally recognized. In order to be of the greatest value in his role as consultant, the endoscopist must be available for these sudden emergencies, and he must be familiar with what constitutes good pediatric practice in the handdling of such problems. In other words, these feeble and delicate infants must be kept warm during all procedures; they cannot be transported to distant operating and x-ray rooms without jeopardizing their slender chance for life; and all manipulations must be gentle.

The endoscopist who is conversant with other disorders of the newborn infant which simulate obstructions in the respiratory tract, is better able to make an accurate diagnosis and institute or recommend appropriate therapy. Beck states, "Two in each one hundred babies refuse to breathe promptly at birth: one-half will never breathe because of permanent injury, and the remainder will live or die according to the measures of resuscitation used." Each case should be decided on its individual merits as to whether or not some endoscopic procedure is warranted.

It should further be remembered that many of our modern mechanical devices for assisting and stimulating respiration are excellent adjuncts to therapy, but they can never replace accurate diagnosis and sound judgment.

1100 East Genesee St.

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LXXXII

SURGERY OF THE PUGILIST NOSE

ALBERT P. SELTZER, M.D., Sc.D.

PHILADELPHIA, PA.

According to the original meaning of the word, present-day competition in the ring is not pugilism. It was applied in the beginning to fist-fighting with bare hands when knuckles were freely used, and infrequently an eye was gourged out. With the adoption of padded gloves, combat in the ring is properly known as boxing. The contest too, if the rules were strictly followed, is not so much to maul one's opponent, as to avoid being struck one's self. But apparently the rules of the same game are sufficiently elastic to allow considerable injury to one or both of the contestants. The result of such injury is seen very commonly in the saddle nose of the professional boxer.

The condition of the fighter's nose as seen by the rhinoplastic surgeon depends upon several factors. Possibly the most important one is the number of times the nose has been fractured, and the parts that have been most severely injured in each succeeding fight. If th nasal injury is given early treatment after each injury, the state of things is quite different than when the results of the injuries have been cumulative.

In general, injuries to the bony nasal arch received in boxing are not apt to be those in which the impact is received on one side or the other. This type of fracture causes the lateral deviation of the entire external nose.

On at least two occasions, the writer has seen fractures of the perpendicular plate of the ethmoid following a severe frontal blow to the face, with penetration of the cribriform plate and consequent injury to the brain substance.

The impact of a heavy blow may cause concussion of the brain of varying degrees. The man falls to the canvas confused or semiconscious so that he may be unable to hear the referee's count. Such forceful blows on the nose may cause edema of the brain or a subdural hemorrhage. When examined in the dressing room after such injury, there may be irregularity of the pupils which is always considered a danger sign. There may be inability to tell the correct

time when shown a watch, although the eyes are open and an effort is made to do so. Such symptoms as scintillating scotoma, ringing in the ears (tinnitus), and even amnesia may occur following severe injury to the fighters nose. The presence of any one or more of these signs determines the end of the contest.

The popular idea of the "glassy eye" of the injured fighter has no real significance in the question of injury. This appearance may be due to a number of factors which mean little more than tears or sweat caused by the intensity of effort in a dusty atmosphere.

The septal cartilage is most apt to be the part of the external nose that undergoes the most frequent and the most severe injury in boxing, and it is this which accounts for the characteristic appearance of the untreated boxer's nose. To some fighters this is a proud possession, a visible sign of their sporting profession.

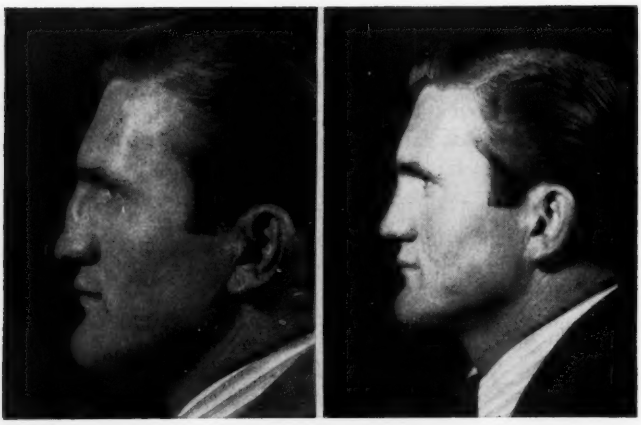
In the beginner, the signs and symptoms of fresh injury to the external nose are bleeding from one or both nostrils. This is the result of injury to the nasal mucosa with tearing of the smaller blood vessels. The amount of the hemorrhage varies from slight or moderate to extreme, and in the latter case the condition may become serious unless prompt attention is given to its treatment.

There is early swelling of the mucosa with edema and possible hematoma, both of which can cause nasal obstruction, which may also be due to the accumulation of blood in the nasal cavities. If the septal cartilage is injured it may deviate to one side or the other filling the passageway and obstructing respiration. This is often evident during the fight in the noisy and difficult breath sounds of the fighters. This may be a snuffing, wheezing, or a whistling sound.

After the first impact of a blow there may be relatively little pain about the nose, unless the nasal lining is severely injured, and there are considerable degrees of swelling.

Soon after such injuries to the nose the loose tissue about the eyes usually becomes infiltrated with blood, giving the familiar appearance of blackening about one or both eyes. This color in the skin may increase during a day or more and then fade out in the course of about ten days.

In boxers who have had as many as 50 or more bouts, there is seldom any notable bleeding from the nose. Repeated injuries have so destroyed the septum and the normal nasal lining, with replacement by dense scar of connective tissue, that vascularity is reduced or the vessel walls are so thickened as to be less vulnerable. This is



not a hard and fast rule, however, as the blood vessels may remain thin and elastic.

In rare cases there may be no external deformity in early injury to the boxer's nose, but on examination with the speculum the various signs are plainly evident.

After repeated fights the tip of the nose always appears rounded and bulbous, as a result of blows on the lower lateral cartilages, by which they may be repeatedly fractured especially about the angle. Just as in all other disorders of this part of the nose, the wings of the tip cartilages are always found to be thin, even with marked nasal injury.

When examined for operation to relieve the marked nasal obstruction of old, repeatedly injured noses, the septal cartilage is found to be both dislocated and to have old multiple fractures. Practically no normal cartilaginous tissue remains, instead it has been replaced by scar tissue that is frequently calcified so that it has the appearance of bone. Injuries to the septum may be associated with drooping or deviation of the tip of the nose, with distortion of the columella.

Operation for repair of the pugilist nose must be treated as major surgery. The patient should be given a very thorough physical examination, to make sure that there is no general physical disorder to contraindicate carrying out the operation on the nose. The presence of severe grade of anemia, or of diabetes particularly should be given consideration and suitable treatment before undertaking the operation. A Wasserman, complete blood count, urinalysis, bleeding time and coagulation are recorded.

In order to assure satisfactory results in reconstructing the nose, front face and profile photographs are made, and a cast of the face is also prepared, upon which a model is molded to be used as a guide.

The operation on an old, badly fractured nose must be done in two or more stages. The finer details of the operation must be suited to the individual case, but in general the customary method of reforming a saddle nose is followed.

The intranasal incision, only one-fourth to one-half inch in length, is made with a Bard-Parker No. 11 blade, and through this incision the skin is undermined widely and completely with scissors. This should be extended in all directions, and carried upward toward the root of the nose to allow sufficient room for the insertion of a graft. The skin of the tip of the nose is then undermined with sharply curved scissors to allow formation of a new tip by means of the angle of the graft to be inserted. In some instances the columella requires extra support and here a cartilage strut is added separately.

The scarred and deformed septum must be dissected very carefully. Both the bony and the cartilaginous parts of the septum may be involved and if the latter has become calcified scar tissue both parts will require chisel and hammer for their removal. The degrees of septal deformity must determine the amount of septal tissue to be removed, and the entire extent of both parts be excised in some cases to assure a clear airway for the nasal passages.

When the new bed for the septal graft has been fully prepared the graft is inserted. This has been sculptured or shaped to correspond with the model made in the beginning. A variety of organic materials has been used successfully for making the graft, including bone, ivory, and cartilage according to the preference of the operator. Among inorganic graft material, vitalium inlays have more recently been employed. This is a light weight metal which does not seem to irritate the tissues in contact with it and so does not cause a foreign body reaction. These inlays are made of very thin strips of well-perforated metal, to make them lighter weight, and to allow the growth of new tissue through as well as about them. They are obtainable in various sizes and shapes to meet almost any need, and can be molded by pressure to suit any contour, although they are firm enough to resist considerable stresses.

An illustration of the usual operation on the pugilist nose can be given by the following case chosen from among a number of prominent fighters, whom the writer has been called upon to treat. They are all quite similar basically, but with detailed differences individually.

This boxer was an Italian who had 31 bouts, 17 of which he had won, and of the 14 lost, he was knocked out in 4. There were injuries by repeated jabs to the nose in every contest. The only treatment at the time of the fights was the application of cold compresses. Inspection showed flattening of the cartilaginous bridge. Internally, there was complete nasal obstruction caused by a thickened and deflected septum. There was no involvement of the nasal bones in this instance. The cartilaginous septum had been comminuted in a way that can best be described as a step-ladder deformity. The condition might be called a fourth degree injury, since it could scarcely be more extreme.

The entire quadrangular cartilage was displaced from the groove in the vomer, and was made up almost entirely of scar tissue which lacked normal covering of mucous membrane.

For the operation the usual method of carrying out a submucous resection was followed as nearly as possible. After removal of the

scarred and thickened cartilaginous septum, a graft of preserved cadaver cartilage was inserted, care being taken that the perichondrium was removed in order to avoid curling of the cartilage which sometimes takes place unless this precaution is observed.

The nose was dressed by using an Asch splint in each nasal passage wrapped in carbowax gauze which was impregnated with sulfathiazole and kephrine hydrochloride. A stent mold was used externally to insure the constant maintenance of the nose in the desired position until union of the parts was complete. The final result of the operation was very good.

An important point for the surgeon before performing a reconstructive operation on the nose of a professional boxer is to have it conclusively understood that his puglistic life must be given up if he wishes to preserve a normal appearing nose, since the operation cannot be repeated with assured success and a nose once reconstructed is particularly vulnerable.

2104 Spruce Street.

LXXXIII

THE RELATION OF TRACHEOTOMY AND LARYNGEAL CARCINOMA TO PULMONARY TUBERCULOSIS

ALVIN D. RUDO, M.D.

BALTIMORE, MD.

AND

DAVID H. JONES, M.D.

NEW YORK, N. Y.

Whether there is a relationship between tracheotomy and laryngeal carcinoma to the activation of pulmonary tuberculosis is a controversial subject. The literature has reported the occurrence of carcinoma of the larynx in tuberculosis patients, but the association is uncommon. Furthermore, where these lesions were found coincidentally no adequate explanation could be given.

Since pulmonary tuberculosis was observed in some of the tracheotomized and radiated laryngeal carcinoma patients at this hospital, it seemed of interest to study this problem further.

During approximately a three year period 43 patients had a tracheotomy performed upon them and 79 patients were diagnosed as having carcinoma of the larynx. This group constituted the subject material of the present investigation. Twenty-nine of the patients with carcinoma were treated by laryngectomy; the remainder received no treatment. A diagnosis of pulmonary tuberculosis was made in ten patients under observation, four of these had no active lesions prior to treatment. In three the underlying pathology was aggravated while only one patient was unaffected by therapy.

The problem of the relation between tracheotomy and the activation of pulmonary tuberculosis has been previously reported. No

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From the Otolaryngologic Section of the Department of Surgery, U. S. Veterans Administration Hospital, Bronx, New York.

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recent studies have been recorded. The concept was considered first before the end of the last century. In 1899, Landouzy[1] asserted that children who had undergone tracheotomy were more prone to develop pulmonary tuberculosis. He based his conclusions on the fact that tracheotomy scars were rarely seen in military recruits. Earlier Trumpp,[2] reported that of 351 tracheotomized patients 64 developed "affections" of the throat, larynx or lung. Pfaundler[1] found that tuberculosis rarely developed after tracheotomy; Wolf,[1] reported four patients with pulmonary tuberculosis in 494 tracheotomies performed in children and Abrahams,[1] three in 101 tracheotomies. Other studies with similar findings for the greatest part involved children, since tracheotomy was apparently more commonly used then in acute diseases of children; adult tracheotomies were a small minority. Tissier[3] also noted four children of ten who had tracheotomies developed active pulmonary tuberculosis. Since the reports are not consistent, the significance of these findings is open to question. The occurrence of tuberculosis in these children could well have been the expected primary infection made easier by "by-passing" the upper respiratory tract filtering and protective mechanism. Rolleston[1] felt that the tracheal phagocytic response was altered by tracheotomy. Blumenfeld[4] regarded irritation by dust and cold air, as well as an increased difficulty to raise sputum by the tracheotomized patient, as etiological factors. The stasis thus produced provided a better avenue for infection by erosion.

Donovan[5] reported the first patient who while wearing a tracheotomy tube for a long time developed pulmonary tuberculosis. Previous reports involved tracheotomy for diphtheria which by the nature of their infectious disease were short-lived fistulae. He felt that there was a definite relationship between unmoistened and unfiltered air entering a tracheotomy aperture to lung infection.

Most authorities feel that tracheotomy accelerates or increases the fatal high rate of active pulmonary tuberculosis. Togvolyi[4] reported 16 patients where conditions were aggravated. In 1917, however, Onody[4] reported laryngeal stenosis following tracheotomy, after which coincidentally pulmonary tuberculosis improved. Similar results were reported in 1924 by Stumberger.[4] Karafiath[4] reported eight patients with advanced tuberculosis in stenotic larynges. He felt that there were two groups of reactions to tracheotomy. The first, a group in which elevated temperature was found, and in which the patients were weakened and had difficulty raising sputum; this is classified as an early reaction. A second group had a delayed reaction, which consisted of an advancement of the pulmonary process; this response may not have been related.

A patient with laryngeal syphilis was reported by Bisi,[6] in which tracheotomy resulted in local and pulmonary activity of tuberculosis.

The literature is barren of information concerning the relation of laryngeal carcinoma and its treatment to the activation of pulmonary tuberculosis. A search of the leading textbooks of otolaryngology and tuberculosis finds no mention of the possible effect of laryngeal cancer on inactive pulmonary tuberculosis[7-13] There are several references to the effect of laryngeal carcinoma upon active pulmonary tuberculosis.

Rokitansky,[19] in the early 19th century, spoke of an antagonism between tuberculosis and cancer, but the observations of Rebattu[14] are at variance with these conclusions. Rebattu presented a number of cases of concomitant Koch bacillus infection and carcinoma of the larynx. It is true that cancer usually appears in people over 40 years of age, and tuberculosis in those in a younger age group. In the group presented here there is a mixture of these concepts.

The patients who have been seen in our clinic are predominantly of two classifications:

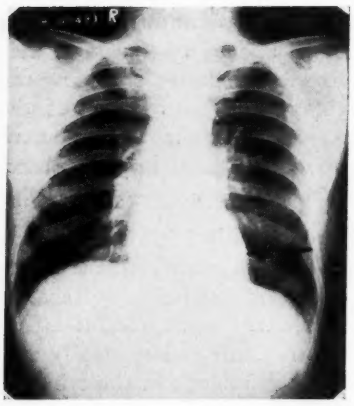
Type A. Tracheotomy and laryngeal cancer apparently awakening inactive or primary complex pulmonary tuberculosis.

Type B. Laryngeal cancer radiation causing effects on active pulmonary infection.

REPORT OF CASES—TYPE A

CASE 1.—C. B., a forty-six year old white male, was admitted to the hospital in April, 1949, for swelling of the neck and hoarseness of 3 weeks' duration. One week before admission a biopsy performed elsewhere from the neck showed epidermoid carcinoma. Physical examination showed bilateral anterior cervical node enlargement, and an extrinsic type of laryngeal growth. Chest x-ray in May showed some widening of the superior mediastinum. After a tracheotomy on May 3, 1949, for a narrowed airway 3500 roentgens were given in three days. X-ray in June showed increased lung markings. Tumor regrowth was seen by July, 1949, and tracheal slough with esophageal fistula appeared soon after. By October there was an infiltrate in the base of the right upper lobe. Sputum examinations were negative for acid-fast bacilli. The patient died on November 17, 1949, and autopsy showed an extrinsic laryngeal cancer with local extension. Caseous bilateral pulmonary tuberculosis was found.

CASE 2.—E. T., a sixty year old white male, noted the onset of sore throat and dysphagia in June, 1943. X-ray treatment was



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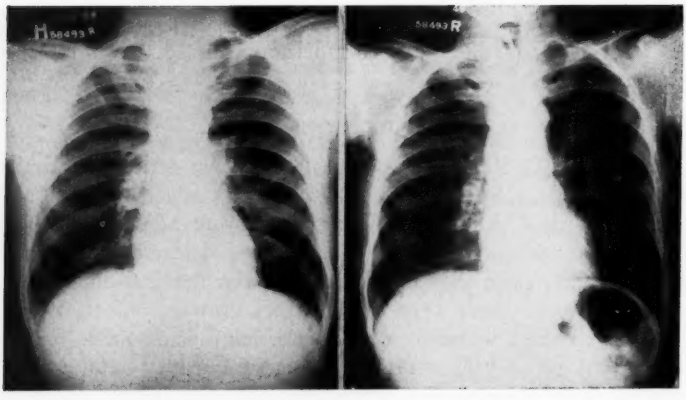


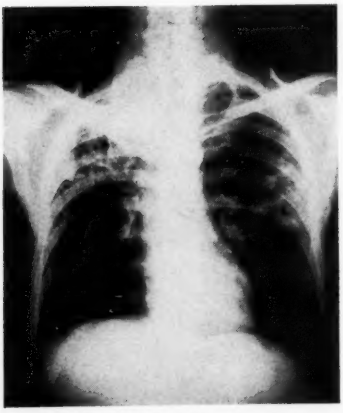
B *C*

Fig. 1, Case 1.

given six months later for laryngeal cancer. He entered our hospital in 1944 for herniotomy. Laryngeal examination and biopsy were negative. Chest x-ray showed a right apical and subapical infiltrate, with adhesions of the pleura. The sputum was negative for acid-fast bacilli. The patient was re-admitted in October, 1947, for dyspnea, as well as laryngeal biopsy. Trachetotomy was performed then, and was closed in July, 1948. The patient returned to the hospital on January 10, 1949, with marked dyspnea and malnutrition. Chest x-ray now revealed a bilateral tuberculous-type infiltrate of both lungs. He expired 35 minutes after his return; no autopsy was obtained.

CASE 3.—J. M., a sixty-two year old white male, weighing 200 pounds with cardiac insufficiency, was transferred to the hospital in February, 1949, because of marked neck swellings, dysphagia, and dysphonia. The neck swelling was present for two years. He presented large irregular bulging neck masses as well as a submucous oro and nasopharyngeal mass. An emergency tracheotomy was performed two days after admission, and artificial respiration administered, following which he was digitalized. Aspiration biopsy was done and reported squamous cell cancer, and 3,900 roentgens were given to the pharynx, and an equal amount to the neck nodes. Chest x-ray showed a pleural reaction one month after tracheotomy, and two sputum examinations were positive on smear for acid-fast bacilli. Culture was negative. The roentgen findings cleared somewhat and he was discharged five months after admission. His tracheotomy wound was closed, the lymph node enlargement had regressed moderately, and dysphagia was relieved. While at home, two months later death occurred. No autopsy was obtained. Review of the chest x-rays showed that an active pulmonary tuberculosis and not carcinoma was responsible for his death.

CASE 4.—E. S., a fifty-two year old white male, had a laryngitis yearly for 24 years, and had noted the onset of severe hoarseness in 1943. He sought medical attention one and one-half years later; cancer was diagnosed and was treated by irradiation (amount unknown). Further roentgen therapy was given in 1947, 6600 roentgens. Tracheotomy was performed the same year for dyspnea. Neck enlargements appeared in 1948, and 5700 roentgens were directed toward them. He was admitted in July, 1948, cachectic and malnourished. Mucopurulent secretions were present around the tracheotomy opening, as well as right cervical nodal enlargement. X-ray showed a right pneumonitis and a left pleuritis. Six months later he had a diffuse linear infiltrate in the right hilar and pericardial regions. A nodule or area of exudate appeared in the left apex.



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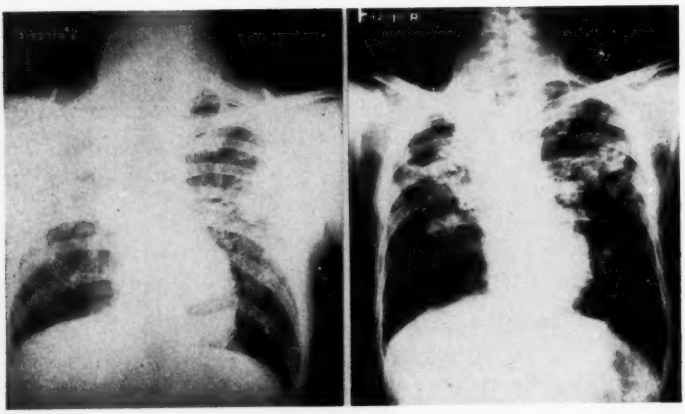

B *C*

Fig. 2, Case 5.

Evidence of recurrence of the laryngeal and neck node cancer was present. Sputum by this time was positive on smear for acid-fast bacilli and elastic tissue. The patient died eight months after admission. Chest x-ray prior to death showed an increase in the size of the mass in the left apex, which might have been metastatic disease. A limited autopsy was granted, but this did not include the chest.

TYPE B

In patients of Type B, the effects of laryngeal radiation on active lung tuberculosis, are as follows:

CASE 5.—J. H., a forty-nine year old white male, was admitted with active bilateral cavitary pulmonary tuberculosis, and carcinoma of the larynx with neck metastasis. X-ray therapy was given, then tracheotomy was performed. Advancement of the pulmonary tuberculosis was rapid, and death followed. Autopsy confirmed the clinical findings.

CASE 6.—J. R., a sixty-three old old white male, was admitted for carcinoma of the tongue with right neck metastasis. X-ray showed a left apical and subapical infiltrate. Sputum examinations were negative for acid-fast bacilli. Hemiglossectomy and tracheotomy were performed. A recurrence was treated with irradiation. Chest x-ray showed a progressive right side disease, resembling tuberculosis. On follow-up examination, sputum and gastric washings remained negative. His follow-up is being continued.

CASE 7.—J. C., a sixty-three year old white male, was admitted for weakness, cough, and dyspnea of two years. Active tuberculosis was found by x-ray and sputum examination. An ulcer on the epiglottis was biopsied and found to be carcinoma. In spite of streptomycin the patient made a rapid exodus in one month. Autopsy confirmed the diagnosis.

CASE 8.—J. M., a fifty-three year old white male, was admitted with the diagnosis of advanced pulmonary tuberculosis, and an extrinsic laryngeal carcinoma which had received 7500 roentgens elsewhere. He died in four days, and no autopsy was obtained. Chest x-ray showed a bilateral pneumothorax, basilar and perihilar infiltrates, right apical infiltrate, and fluid in the left costophrenic angle.

CASE 9.—J. C., a seventy-four year old white male, was admitted with the history of hoarseness and dysphagia of eight months duration, with a 58 pound weight loss. Active bilateral apical tuberculosis (gastric washings positive) and extrinsic laryngeal carcinoma were present. A total of 6600 roentgens were given to the larynx.

Follow-up studies have shown no advancement in the tuberculous status after six months (by x-ray and clinical appearance). The cancer has regressed markedly.

CASE 10.—J. K., a fifty-two year old white male, was admitted with signs of active pulmonary tuberculosis. Three months later, in another hospital, he was treated by radiotherapy for an extrinsic laryngeal carcinoma. Progression of the tuberculosis has been noted in spite of therapy with streptomycin, paraminosalicylic acid, etc. Advancement of the cancer is also seen at present.

Eggston[10] states that "tuberculosis of the larynx is not infrequently associated with an epidermoid carcinoma." He feels that tuberculosis always precedes the carcinomatous "degeneration." However, this has been refuted in the past by such investigators as Rebattu.[14] None of our autopsied patients showed evidence of the presence of laryngeal tuberculosis. Another observer[15] pointed out, "the idea that tuberculosis is a precursor of cancer cannot be supported, especially in coexistence in the larynx, since carcinoma and tuberculosis are rarely associated elsewhere."

Is there a deleterious effect of laryngeal cancer on pulmonary tuberculosis? From these case studies presented herein the authors are inclined to answer in the affirmative. Clement,[16] in observing four patients, concluded that cancer had a stimulating effect on local and generalized tuberculosis. In fact, one author postulated that if more autopsies were performed in laryngeal cancer, more active tuberculosis would be discovered, and vice versa. A reversed phenomenon has been seen in experimentally induced tuberculosis, where cancer of the same organ then regressed.

It has been stated that in cases of combined pulmonary tuberculosis and laryngeal cancer, the lung process seems to be the one that is the cause of death. It is, therefore, of paramount importance that the tuberculous process be given active treatment immediately after it is discovered. A good general body improvement and build-up is necessary when the carcinoma is attacked. Many authors fear surgery because even a biopsy has been known to activate tuberculosis.[14, 17]

The nature of the probable relationship of the cancer is obscure. Terracol[15] feels that the malignant tumor does not appear to favor the development of the bacillary lesion, and vice versa. However, he does point out that cancer should be suspected and intricately searched for in ulcerated tuberculous larynges. From this study it can be added that pulmonary tuberculosis should be sought for in patients with laryngeal carcinoma.

A consideration of the present concepts of the pathogenesis of tuberculosis may give some clues to the relationship we seek to explain. Among phthisologists it has long been argued whether adult-type tuberculosis is of endogenous or exogenous origin. The fact that proof is most dependant upon experimental investigation makes this an almost insoluble problem. Guinea pigs are too susceptible to human and bovine and rabbits to bovine organisms, to be of use. However, exogenous superinfection suggests itself in persons primarily infected, and who are in close and prolonged contact with heavy bacillary sources.[13] Bronchogenic tuberculosis follows primary infection in a short time in young adults, but a long interval is present in middle aged and elderly people. Many authors cite the prolonged exposure in the case of married people and the incidence of partner-activation is reported all the way from 2.2% to 58%. Repeated exposure in children is generally regarded as a source of reactivation or reinfection.

Virulence of tuberculosis bacilli is still an unknown and inadequately measureable quantity. As for many other infections, a depression of "resistance" can render one susceptible to infection from without, or to endogenous metastatic reinfection. The effects of malnutrition to the "resistance" have never been definitely established in controlled experiment. Are there any special dietary factors involved? Tuberculosis is often seen, it is true, in the inanition of carcinoma of the stomach and esophagus. Is it that certain body resistance factors are injured?

Exercise certainly has deleterious effects on this infection. Mental factors can be considered too, as also such diseases as angina and peptic ulcer.

Intercurrent infection is assumed to play a major role. This certainly could be a factor in tracheotomy cases where secondary bronchitis and susceptibility to upper respiratory diseases is produced. Alcohol is blamed for its inhibiting effect on phagocytosis. Several of the patients presented had long histories of the use of alcohol.

Pathogenically, trauma has a definite place in the pathologic processes of activating and progressing tuberculosis. From this investigation one must postulate that the effect of radiation therapy is also a factor. It is known that radiation therapy of the lung for any reason can easily activate latent or inactive tuberculosis.[7] Whether localized laryngeal radiation has a direct or more likely an indirect effect is, of course, purely conjectural.

Dworetsky[21] classifies pulmonary tuberculosis into two main groups: (1) Bronchogenic or bronchopulmonary. (2) Hematogenous.

In the bronchogenic or bronchopulmonary form, the lungs are invaded by the tubercle bacillus through the air passages and the pulmonary lesion spreads to other parts of the lungs mainly by way of the bronchial route. This form occurs in about 80% of all cases of pulmonary tuberculosis, exudative, caseous, fibrocavernous, and productive types.

In hematogenous forms the tubercle bacilli spread through the lungs and other organs by way of the blood stream. Such invasion of pulmonary tissues occurs in about 20%. Here tuberculosis may be mild or severe depending on many factors. Rubin[22] states that when tubercle bacilli invade the blood stream there may be generalized miliary tuberculosis which may be severe or there may be complete absence of symptoms.

The severity of symptoms depends upon the number and virulence of the bacilli, how often they invade the blood stream, the site of entrance into the blood stream and the sensitivity of the body to tuberculotoxins. At the beginning, pulmonary tuberculosis may start either as bronchogenic or hematogenous. It very often becomes mixed.

Edgar Mayer[23] states that with our modern refinements in x-ray technique we now see earlier signs of pulmonary tuberculosis. The type of lesion now seen is different from the old form and can only be seen by x-ray. The occurence of pulmonary tuberculosis found in the patients described in this reference may have been infected in childhood but the chronic pulmonary disease begins only in adult age from an already old apical lesion, its evolution being slow and insidious. Parke, Loftus, and Bishop[24] stated that patients who have had thoracic surgical operations have a wider spread of tuberculosis due to over-medication preoperatively with loss of cough reflex. This might occur following laryngeal and neck surgery for malignancy.

CONCLUSIONS

1. Ten cases of pulmonary tuberculosis are presented; they occurred in a group of 79 laryngeal cancer patients and 43 tracheotomies.

2. There is a cause and effect relationship between tracheotomy and irradiated laryngeal cancer and the activation and accentuation of pulmonary tuberculosis.

3. It is recommended that treatment of patients with both lesions should be directed toward the tuberculosis process immediately.

4. It is recommended that laryngeal cancer patients be closely watched before and after treatment for signs of pulmonary tuberculosis.

5. It is postulated that many patients who have severed connections with their treatment centers have succumbed to pulmonary tuberculosis, the death being assigned by the family physician to carcinoma.

6. The literature has advanced the theory that wearing of a cannula causes an unnatural inspiration of air which has not been previously warmed, moistened, and filtered. It is further asserted that cold and dust-laden air may damage the lower air passages and lungs, and contribute to the development of bronchopulmonary tuberculosis.

CHARLES AND READ STREETS.

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LXXXIV

TREATMENT OF OTOGENOUS TEMPORAL LOBE ABSCESS

HANS BRUNNER, M.D.

NEWARK, N. J.

The majority of otologists share the view that the treatment of otogenous brain abscess belongs in the realm of the otolaryngologist who is adequately trained to eliminate the infection of the ear and of the brain in one surgical procedure. Exceptions are perhaps instances in which the brain abscess is caused by an acute otitis and in which the infection of the ear has subsided when the brain abscess causes the first symptoms. In all other instances the otolaryngological approach is adequate. The strictly neurosurgical methods may drain the abscess very well; yet they do not eliminate the primary source of infection in the ear (or sinus) and, thus, render two or more surgical procedures necessary in order to take care of the entire area of infection. This is well proven by the following case:

CASE 1.—S. Y., 14 years, white, female. When she was a child she had measles and scarlet fever which were followed by a bilateral otitis media. Three weeks ago she caught cold and complained of slight earache on the right side. There was a slight amount of purulent discharge from both ears, chiefly on the right side and the temperature rose to 104. Sulfathiazol was administered. During the next 10 days the temperature varied between normal and 102. At this time there was a profuse purulent discharge from the right ear which relieved her headache and earache slightly. On February 7, 1947, she was brought into a hospital where she was given penicillin, intramuscularly and intravenously, and sulfonamides. The neck was stiff and painful; in the spinal fluid there were 1390 cells (67 polynuclear cells) and Gram positive cocci, morphologically identical with pneumococci. The diagnosis of a pneumococcic meningitis was made and daily spinal punctures were performed. On February 12, there was a convulsion immediately after intravenous injection of protein. On February 13, there was another immediately after a spinal puncture. During the attack the patient's head and eyes turned to the left; the left upper and lower extremities jerked, especially the arm. After the convulsion the abdominal reflexes were absent on the left, and brisk on the right side. Her headache was localized to the right frontal area. On February 14 she was brought into the hospital for eventual surgery.

At admission, she was a lethargic, extremely pale girl. She wore a hearing aid on the left mastoid. There was marked dehydration and weight loss, the pulse rate was 100, the temperature 101.4. She cooperated fairly well if the voice was raised. There was a marked bilateral papilledema with tortuous vessels, a weakness of the left abducens, a paresis of the left facial nerve, a weakness of left arm and leg and a marked hyperesthesia over the entire body. The neck was stiff and there was pain on elevation of the head, but there were no pyramidal symptoms. There was a left homonymous hemianopsia. In the spinal fluid there

were 119 cells and the spinal pressure was 260 mm of water. The blood hemoglobin was 10.5, there were 4,000,000 erythrocytes and 16800 leucocytes (polynuclear leucocytes 80%, lymphocytes 20%.) Under local anesthesia an incision for temporal decompression was made by the neurosurgeon. Four punctures of the brain, directing the needle downward and backward were performed. Fifty cc of sterile pus were removed and 1,000,000 units of penicillin were injected. On February 15 neither the drowsiness nor the meningeal symptoms had changed. The temperature was 102.8 and the pulse rate 100. The left ear drum was totally destroyed; on the right side there was a marked sagging of the superior and posterior wall of the external canal, the tympanic cavity was filled with fetid pus, the tip of the mastoid was not edematous. There was no nystagmus. The blood hemoglobin was 11.0, there were 3,200,000 erythrocytes and 16000 leucocytes. An incomplete mastoidectomy was performed on the right side. The mastoid process was sclerotic. In the antrum there were discolored granulations and a small amount of pus. The bone of the mastoid was traversed by channels filled with granulations. In the tympanic cavity cholesteatoma and granulations were discovered. The matrix of the cholesteatoma was removed, to a great extent. There was a defect in the tegmen of the antrum. The entire tegmen was removed. The dura was bulging, hyperemic and covered with fibrin. A nick incision was made. Four punctures of the brain did not yield pus. A cross incision was made in the dura which was adherent to the cerebral cortex due to fibrinous exudate. The four flaps were reflected. The cortex was cyanotic and edematous and formed immediately a herniation the size of a walnut. On February 16 the temperature was 99.8 and the pulse rate 86. Both optic discs were elevated, edges blurred and there was an edema of the surrounding retina. The papilledema was 3D on the right side, 2D on the left side. In the blood hemoglobin was 10.5, there were 3,100,000 erythrocytes, 16,200 leucocytes (lymphocytes 20%, polynuclear cells 78%, eosinophiles 2%). She felt well. On February 17, the temperature was 99.8 and the pulse rate 92. In the blood there were 8,500 leucocytes (lymphocytes 25%, monocytes 3%, polynuclear cells 72%). 1,000,000 units of penicillin were administered daily. On February 19, the spinal fluid was cloudy and sterile. In the blood the hemoglobin was 10.8, there were 3,300,000 erythrocytes, and 9500 leucocytes. On February 20, the temperature was 98.4 and the pulse rate 88. There were 4,000,000 erythrocytes and 7100 leucocytes. The patient was dismissed on March 7 after having received a total of 2100 grains of sulfadiazine and 12,000,000 units of penicillin (70,000 units intrathecally). On March 18, the patient was completely well. The brain herniation filled up the entire radical mastoid cavity and was covered with granulations. X-rays were applied to the herniation which gradually was changed to connective tissue and slightly diminished in size.

This was a typical case of otogenous temporal lobe abscess caused by a cholesteatoma of the right tympanic cavity and entering the manifest phase due to an acute flare-up of the cholesteatoma. The brain abscess was drained by a neurosurgeon through the temporal squama. The tympanic cavity was let alone. Despite the proper drainage of the abscess the patient remained drowsy, meningeal symptoms and fever were unchanged on the day following the operation. For this reason a radical mastoid operation was immediately performed and an external pachymeningitis and an edema of the temporal lobe were discovered. On the day following this operation the drowsiness and the fever had decreased and two days following the ear operation the temperature was normal. The patient was dismissed with a brain herniation filling the entire radical cavity. The

herniation was gradually replaced by granulations and finally by connective tissue.

It is evident that the favorable result which was achieved in this case by two operations could have been accomplished as well by one surgical procedure. And even with the assumption that the encephalitis causing the brain edema may have been eventually relieved by antibiotics, the active cholesteatoma of the tympanic cavity would have required an operation under any circumstances.

The reason for discarding the evident advantage of the otolaryngological procedure is supposed to be the hazard involved in operations through an infected field. The writer has on several occasions[1, 2] emphasized that this statement should not be generalized. While the normal leptomeninges may be highly susceptible to infections, their susceptibility is apparently diminished when on one occasion they have defied an infection by the formation of scars. This is exactly what happens in otogenous brain abscess since the formation of a brain abscess is usually preceded by a localized meningitis. Furthermore, the brain substance has apparently a fair resistance to ordinary bacterial infection which is proven by the rarity of brain abcess after unsuccessful punctures of the human brain and by experiments in animals. Understanding the responsibility which is involved in a statement of this type, the writer ventures to say that the hazard of draining a brain abscess through an infected field has been exaggerated prior to the advent of the antibiotics and is definitely exaggerated after. In consequence of this view the writer has suggested the use of the socalled open method in an infected field. The open method includes briefly the following procedures: cross incision of the dura, introduction of a cannula into the abscess and marsupialization of the abscess.[1, 2] The method is not original with the writer; it has been used by several neurosurgeons.[3] However, it has been used exclusively in a sterile field and there has given good results. Since the operation implies a wide opening of the meningeal spaces and an extensive injury to the brain it was considered a strictly sterile procedure although, from the theoretical point of view, the sterility of the surgical field can scarcely be maintained when the contents of the abscess cavity escape.

The excellent exposure on one hand, the radical drainage of the abscess on the other hand, furnished by the open method, have given rise to the idea of combining this method with the operation upon the ear. Naturally the original method which is adjusted to the approach through a sterile field, had to be modified according to the conditions in the ear. The most important modification concerned the management of the meninges. In the original method the cross

incision of the dura results in four flaps which are reflected. This is not always feasible when the operation is performed through the ear because in this approach an infected area is traversed. Consequently the meninges in this area frequently show active or residual inflammation granulations on the outside of the dura and adhesions between dura and cerebral cortex. While the granulations on the outside of the dura do not interfere seriously with the employment of the "open" method, the adhesions do, because under no circumstances should the adhesions be torn. The writer believes that a fatal meningitis in one case of temporal lobe abscess in which the open method had been employed, was caused by tearing the meningeal adhesions. For this reason, a preparation of the four dural flaps is not always feasible at the base of the temporal lobe and, therefore, the technique can not always be applied if the operation is performed in the infected field. The type of modifications which has to be applied in these instances, is well demonstrated by the two following cases.

CASE 2.—A. K., 60 years, white female. She had one month ago a quinsy associated with fever up to 102.0 A peritonsillar abscess was drained and she was treated with penicillin. After one week she was cured. Following this episode she complained of buzzing in the left ear and intermittent moderately severe earache, characterized by throbbing pain. A few days later there was a discharge from the left ear, serous first and purulent later. At that time she still thought she had fever but there were no chills, nausea or vomiting. She was brought to the hospital to be treated for pyelitis, arteriosclerotic heart disease and auricular fibrillation.

At admission, on April 27, 1948 the temperature was 98.4 and the pulse rate 88; on the evening of the same day the temperature was 102.8 and the pulse rate 100. The right ear was normal. On the left side the drum was thickened, the landmarks indistinct; a perforation could not be visualized but there was pus in the external canal. The left tonsil was inflamed. Bloodcultures of 5 colonies of pneumococci per 1 cc of blood were obtained. It was not possible to repeat this finding. There were 4,600,000 erythrocytes and 31,500 leucocytes. She received 40,000 units of penicillin every 3 hours. On April 28, the sedimentation rate was 27 mm in 2 hours. In the smear from the tonsils staphylococcus albus was found. On April 30, there were 3,500,000 erythrocytes and 13,250 leucocytes (8% juvenile forms, 63% polynuclear cells, 1% eosinophiles, 28% lymphocytes) in the blood. In the pus from the ear Gram positive diplococci and staphylococcus albus were discovered. After a few days she was dismissed since her temperature was normal and the ear was apparently cured.

Two days later, on May 13, her sensorium was suddenly disturbed. She recognized the members of her family only intermittently, she asked for her fur when she really wanted her tooth prosthesis and so on. On May 15, she had the following seizure: her eyeballs went up, her arms went limp at her sides. A gurgling noise was heard in her throat; unconsciousness followed for about 10 minutes. On returning to bed she vomited. Confusion followed. She suffered from diarrhea and her stomach was quite distended.

On admission, on May 15, 1948, she was confused and disorientated. Her temperature (orally) was 99.6 and her pulse rate was 80. The left pupil was larger than the right, the motility was normal. There were no other neurological

symptoms except the aphasia. Whatever was shown to her, she called "powder." (Just before the seizure she had asked for powder for her face.) The heart was fibrillating and there was an abundant secretion from the left ear. For her endocarditis, dicumarol was administered. In the blood there were 3,980,000 erythrocytes and 14,700 leucocytes 8% juvenile forms, 71% polynuclear cells, 20% lymphocytes, 1% monocytes). Penicillin was administered.

On May 18, there were 4,680,000 erythrocytes and 15,950 leucocytes (8% juvenile forms, 50% polynuclear cells, 8% eosinophiles and 21% lymphocytes and 13% monocytes) in the blood. On May 21, there was an increasing eosinophilia (perhaps due to dicumarol). On May 22, there were 4,350,000 erythrocytes and 8,000 leucocytes (15% juvenile forms, 42% polynuclear cells, 18% eosinophiles, 22% lymphocytes and 3% monocytes) in the blood. On May 24, the reflexes of Babinski, Oppenheim and Gordon were positive on the right side; there was a clonus on both sides, on the right side more than on the left and in Romberg's position she fell to the right. Spinal fluid was clear and the pressure was normal. In the spinal fluid there was a normal omunt of cells, Pandy++, glucose 36, protein 3 gm, Kahn negative

When I saw her for the first time on May 24, she did not speak at all. If something was shown to her she smiled and mumbled something which was not understandable. If she was asked to perform a movement she did not understand but she mumbled something like "That's so—that it." It was possible to persuade her to repeat figures; she did it from 1 to 6, then she suddenly stopped, smiled and mumbled "That's so." If she was asked by writing to do something she could not read. Both malar processus were tender; but there was no tenderness of the atlanto-occipital membrane and the eye balls. There was a slight edema and tenderness of the left mastoid process. There was no pus in the left external canal. The left drum was gray, the landmarks obscured and there was a sagging of the superior and posterior wall of the external canal. The posterior quadrant of drum was slightly bulging. The right ear was normal. There was a slight swelling of the left tonsil and of the left tonsillar pillars. She was not stone deaf on the left side. There was a horizontal and rotatory nystagmus of I. degree to the left. The diagnosis of a temporal lobe abscess on the left side was made.

On May 25, an incision was made in the area of the left mastoid. There was some mucopus in the antrum and in the tip of the mastoid. In the smear pneumococci were found, in the culture only staphylococcus albus and no anaerobic bacteria. The mastoid was diploetic. The tegmen antri was necrotic and was entirely removed. There was a great amount of granulation on the dura. A part of the posterior wall of the external canal and of the lateral wall of the antrum were removed, but the bridge was not taken off. The horizontal semicircular canal and incus were normal. In the area of the sulcus digastricus there was a fistula leading toward the inferior knee of the sinus. In that area the sinus was covered by pus. For this reason, the superior angle of the petrous bone was removed and the sinus was exposed from the jugular bulb up beyond the superior knee. The sinus was thickened and covered with granulations. Incision of the sinus revealed granulations, connective tissue and small black thrombi in the sinus, but no blood and no frank pus. Now a perpendicular incision, 4 cm in length, was made above the auricle. The temporal muscle was pushed aside and the normal dura was exposed at the convexity of the temporal lobe in a size of one-half of a dollar. A cross incision was made in the inflamed dura at the base of the temporal lobe . The posterior two flaps could be reflected; the anterior flaps were adherent to the cortex by adhesions and were left in place. The cerebral cortex was pale, edematous and slightly bulging. The veins of the pia were not dilated. A puncture of the brain failed because below the cortex the cannula met with a resistance which could not be pierced by the cannula without using undue pressure. This was the thick capsule of the abscess which had to be incised by a scalpel. Now the cannula entered the abscess and from a depth of about 3 cm approximately 20 cc of a cloudy, sanguino-

lent fluid escaped mixed with viscid mucus and inspissated pus. In the smear there were many polynuclear cells and monocytes and a small amount of Gram negative cocci. By culture neither aerobic nor anaerobic bacilli were obtained. When the pus had escaped there was a good pulsation of the brain and a minimal herniation of the brain. With the electric knife a part of the cerebral cortex together with the capsule of the brain, about the size of a one cent piece, were excised. Further punctures did not reveal any pus.

Microscopic examination: The cerebral cortex is poor in ganglion cells. The spaces of Virchow-Robin are filled with lymphocytes and plasma cells. There are no polynuclear cells. The capsule of the abscess consists of hyaline connective tissue and a small amount of capillaries, the walls of which are slightly infiltrated. Toward the abscess cavity the connective tissue is replaced by granulation tissue which shows an intense infiltration.

On May 29 the aphasia was slightly improved. She understood a few orders, she also recognized some of her relatives but could not call them by name. There were no meningeal symptoms. There was a small herniation of brain which did not extend into the antrum. There was no pus. On June 1, there were 3,800,000 erythrocytes and 11,500 leucocytes (3% juvenile forms, 57% polynuclear cells, 2% eosinophiles, 37% lymphocytes, 1% monocytes) in the blood. On June 2, there was a slight paresis of the facial nerve on the right side. There was no Babinski reflex, and the ankle clonus was well sustained on the right side, not sustained on the left side. Yesterday one reading showed a pulse rate of 62. There was no papilledema. In the blood there were 3,660,000 erythrocytes and 10,200 leucocytes (5% juvenile forms, 44% polynuclear cells, 1% eosinophiles, 50% lymphocytes). On June 5, the aphasia was improved, but when she became tired her aphasia was worse. The brain herniation was slightly increased filling a part of the antrum, and was covered with granulations. On June 10, she was out of bed, but her aphasia had not changed. She could eat by herself but she had trouble with coordination in the right hand. Thus, she spilled food and liquids which made her angry. The motor force was equal in both hands.

On June 12 she continued to receive 1,000,000 units of penicillin daily. There were occasionally slight rises of temperature, the blood finding was normal and the aphasia was improved. There was a cortical paresis of the right facial nerve, the abdominal reflexes were diminished on the right side and there was an ankle clonus on both sides. On June 19, her temperature was normal. She felt well and there was a slight degree of euphoria. She insisted on going home. Her spontaneous speech was normal and there was no paraphasia. If she was told to memorize the names of the months or to count she did not understand. If, however, the word January or one was spoken to her, she continued promptly. Simple orders were promptly executed. Her memory was not quite normal. When she was asked, "How many daughters do you have?" She said "Three." (She has only two and gave the correct names of both of them.) There was some perseveration. She recognized the physicians and the nurses but did not know their names. If the names were told her she repeated them correctly. She did not find the correct words for colors. She could not read and did not make any effort to do it. A pencil was given her upside down, and she was told to write. She laughed because she believed to be fooled. She turned the pencil in the right position and wrote correctly her name and her address. There was a tremor of the right hand but there was no apraxia. She could eat without assistance, but she could not walk without assistance because she complained of dizziness. There was a slight hyperalgesia all over the body, the motor force of the right arm and leg was slightly diminished, abdominal reflexes were diminished on both sides. There was no dermographism, no nystagmus, no facial paresis. Corneal reflexes were positive on both sides. The tendon reflexes of the left leg were slightly exaggerated, there was an ankle clonus on the left side, Oppenheim's and Bakinski's reflexes were positive on both sides. There was no ataxia. The mastoid was well granulated and there

was no herniation of brain. On July 23, she felt well and the aphasia had disappeared to a great extent. On July 24, there was a small amount of secretion from the tympanic cavity and there were granulations in the area of the Eustachian tube. In the mastoid area there was a deep depression, epithelized to a great extent. There were no rhythmic pulsations in that area. She was depressed and complained of tinnitus on both sides. (Prior to the operation there was tinnitus on the right side only.) Otherwise her condition was good.

In this case there was an acute otitis on the left side which ran a course typical of an infection with pneumococcus III. The ear infection caused a sinus thrombosis which, at the time of the operation, was cured to a great extent by the administration of antibiotics. It is likely that the sinus thrombosis had been active during her first stay in the hospital when she was treated for pyelitis and heart fibrillations. The correct diagnosis had not been made at that time, yet a spontaneous cure of the sinus thrombosis was achieved by the antibiotics which were actually given for her heart and kidney conditions.

In addition, she developed a temporal lobe abscess which, at first, was believed to be a vascular disease of the brain. This error was probably caused by the course of the otitis which differed considerably from the course of a common acute otitis. The drum did not exhibit the signs of an acute inflammation at any time, there was abundant discharge at one time and practically no discharge from the tympanic cavity at another time. For this reason, the otitis was considered to be harmless although it is just this finding which points to a "mucosus otitis" with all the hazards involved.

At operation the brain abscess was not more than 8 to 9 weeks old since that was the duration of the acute otitis. It is certain that the duration of the brain abscess was much shorter. If the day on which the patient noticed her first and only seizure is considered to indicate the initial phase of the abscess—this is by no means a far-fetched assumption—the duration of the abscess would have been about 2 weeks. In this time the infection had produced a thick capsule at the base of the temporal lobe and the abscess had entered the quiescent phase.

It seems rather strange to refer to a quiescent phase in a case who presented symptoms as prominent as case 2. However, the various phases of brain abscess do not depend on focal symptoms but on systemic symptoms. Assuming that the abscess of case 2 would have been located in the right temporal lobe there would have been no symptoms of aphasia and the abscess would have been in better agreement with the usual concept of a quiescent phase. In other words, the symptoms of case 2 had no relationship to the "phase" of the abscess but to its localization in the left temporal lobe. For this reason, the symptoms of aphasia like all other

focal symptoms should be discarded so far as the diagnosis of certain phases of brain abscess is concerned. In this respect only the systemic symptoms and the surgical findings are indicative. The systemic symptoms were poor in case 2 at the time of operation; in fact, there was only the irregular fever which pointed to an intracranial complication. Of great importance were the surgical findings. The cerebral cortex was pale, only slightly bulging, the veins of the pia were normal, there were no bacteria (also not anaerobics) in the pus and there was no herniation of the brain after surgical drainage. These findings definitely indicate the quiescent phase of the abscess. In this case the operation was performed on a brain abscess in the quiescent phase and caused by an acute otitis, probably due to an infection with pneumococcus III.

The technic used in this case was close to the original method, but it was not exactly the original method. The cross incision was made in an inflamed dura while in the original method the incision is made in a normal dura. It was possible to reflect the posterior two flaps but the two anterior flaps were fastened to the cerebral cortex by firm adhesions. For this reason, the two anterior flaps were left in place and the cerebral cortex together with the casule of the abscess were excised in an area where the reflection of the dura flaps had been feasible. The patient was observed for 5 months only. She made good progress and there was no secondary herniation of brain.

Case 3.—R. W., age 51, white, female. She had scarlet fever and measles in childhood. Ever since she has suffered from a discharge from both ears. She has never had an ear treatment for a sufficient period of time. Nevertheless, the discharge ceased definitely in the right ear. The left ear has been discharging for the last 7 years. Since 1938 she has had many attacks of turning dizziness but never headaches. Her hearing has markedly diminished on both sides for a long period of time. Since October 1946, the discharge from the left ear suddenly ceased without causing headaches. The dizzy spells continued. On December 24, 1946, she suddenly noticed severe pain in the left ear, there was severe dizziness, particularly when she moved her head, severe headache and a gradually increasing dullness of her sensorium. Some time she could not remember that she was brought into the hospital and that two operations were performed. This, however, was not only due to the dullness of the sensorium but very likely to a retrograde amnesia.

On admission, on Dcember 28, 1946, there were 3,700,000 erythrocytes and 28,650 leucocytes in the blood and the sedimentation rate was 27 mm. On December 30, there was a slight paresis of the left facial nerve. She was very dizzy, but there was no rigidity of the neck and the eye fundi were normal. I saw her on December 31, for the first time. There was a total destruction of the right ear drum, but there was no discharge. There was a foul smelling discharge from the left ear, a marked sagging of the superior and posterior wall of the external canal. The tympanic cavity was filled with cholesteatoma. There was no fistula symptom and the left mastoid was tender. Weber was not lateralized and there were no cerebellar symptoms. The sensorium was slightly dull

There was a paresis of the mouth branch of the left facial nerve and a horizontal and rotatory nystagmus of III degree to the right. There was tenderness on the left side of the face, but there was no hyperalgesia, no rigidity of the neck and the corneal reflexes were normal. The following tentative diagnosis was made: a.) acute exacerbation of a cholesteatoma on the left side, b.) diffuse purulent labyrinthitis(?), c.) mastoiditis, d.) incipient meningitis. Since the labyrinthine symptoms were very acute, penicillin, hypertonic glucose and hydrogen peroxide into the ear were administered.

On January 1, 1947, the spinal fluid was normal except for a +Pandy. On January 2, earache, headache and nystagmus were diminished. There were 3,730,000 erythrocytes and 11,100 leucocytes (poly nuclear cells 59%, juvenile forms 9%, eosinophiles 2%, lymphocytes 29%, monocytes 1%) in the blood. On January 5, the spinal fluid was normal except for a +Pandy. There was marked drowsiness and dryness of the tongue. The facial paresis was improved but there was a horizontal and rotatory nystagmus of III degree to the right, tenderness of the left malar bone, tenderness of the atlanto-occipital membrane and of the left mastoid, but there was no tenderness of the eye balls, no dermographism, no hyperalgesia. The fistula test was uncertain and there was perhaps some excitability of the left labyrinth. Due to drowsiness, the cooperation of the patient was poor. Now the diagnosis was made of a.) cholesteatoma, b.) secondary serous labyrinthitis, c.) exteradural abscess of middle and perhaps posterior cranial fossa. Immediate surgery was suggested.

At operation, retention of pus was discovered in the antrum, containing staphylococcus haemolyticus. The antrum was enlarged and a great amount of watery, non-fetid pus, containing some mucus, escaped from the area of the Eustachian tube showing rhythmic pulsations. The antrum was filled with cholesteatoma and the tegmen antri was absent to a great extent. A radical mastoid operation was performed and a great amount of cholesteatoma was removed. An oval fistula, 5 mm in length, was discovered on the horizontal semicircular canal. The fistula was filled by the hyperemic endosteum. The horizontal portion of the facial canal was invaded by granulations. The dura of the middle fossa was covered by granulations which, on some places, were changed into connective tissue. The granulations extended toward the apex of the petrous bone as well as to the major wing of the sphenoid. In these areas it was not possible to reach normal dura. The superior angle of the petrous bone was removed. The sinus was apparently normal. The skin incision was left open. On January 7, the spinal fluid was normal except a +Pandy. The nystagmus was less marked. On the following days the temperature was slightly elevated, the pulse rate varied between 60 and 100. On January 8, it was difficult to wake her. There was a paraphasia which was not noticed yesterday. There were no distinct meningeal symptoms. The tendon reflexes were exaggerated on the right side, the abdominal reflexes were diminished on both sides. Signs of Babinski and Oppenheim were indistinct on the right side. The motility on the right side was restricted. There was no nystagmus and the facial paresis on the left side was diminished. The diagnosis of temporal lobe abscess on the left side was made. On January 10, there were 4,050,000 erythrocytes and 13,550 leucocytes (juvenile forms 15%, polynuclear cells 56%, lymphocytes 28%, monocytes 1%) in the blood, the color index was 47. The dullness of the sensorium grew worse, there was incontinentia alvi et urinae and the temperature rose to 101 while the pulse rate fell to 60.

An operation was performed on January 14 under light ether anesthesia. More dura was exposed toward the temporal squama and the sphenoid wing. The dura which had been exposed at the first operation, was covered by a thick layer of granulations, but there were no necroses. A nick incision was made at the base of the temporal lobe. Punctures were performed in the directions forward, mesialward and upward until a depth of 6 cm, without releasing pus. Puncture

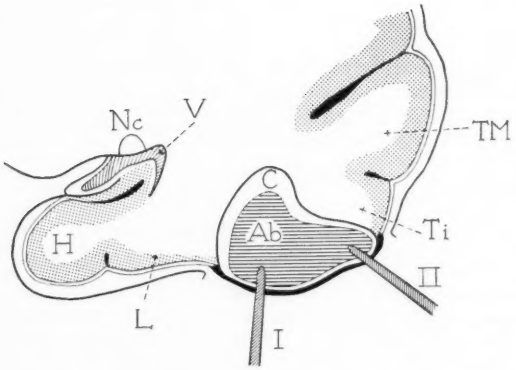

Fig. 1, Case 3.—*Ab*, Abscess cavity; *C*, Capsule of abscess; *Ti*, Inferior temporal gyrus; *TM*, Mesial temporal gyrus; *L*, Lingual gyrus; *H*, Gyrus hippocampi; *Nc*, Caudate nucleus; *V*, Inferior horn of ventricle; *I* and *II*, Cannula introduced into the abscess cavity. The tissue between the two cannulas was excised.

in the direction toward the inferior horn meets with a resistance apparently due to a thick capsule. After perforation of the capsule about 5 cc of fluid escaped, similar to a cloudy spinal fluid. The suspicion was aroused that the cannula had entered the inferior horn. The cannula was left in place and after one or two minutes creamy pus escaped through the cannula indicating that the cannula had entered the abscess cavity and not the inferior horn. Approximately 20 to 25 cc of creamy, non-fetid pus, containing staphylococcus albus, were aspirated. The nick incision was enlarged to a cros incision and an attempt was made to prepare 4 dura flaps. This was not possible because of the firm adhesions which fastened the dura to the brain. For this reason a second incision was made through the dura at the convexity of the temporal lobe where the pachymeningitis was less marked. A second cannula was introduced through the second incision; the capsule was again perforated until the cannula met with the first cannula within the abscess cavity (Fig. 1). Both cannulae were left in place. An attempt to prepare the dura flaps at the convexity likewise failed because of firm adhesions. this attempt resulted in a considerable hemorrhage from dilated pia veins. The hemorrhage was stopped by a tampon soaked with hydrogen peroxide. The tissue between the two cannulae, consisting of inflamed dura, plus cerebral cortex, plus abscess capsule was excised creating a hole the size of a dime. No drainage of the abscess was made, however, the cavity in the mastoid was drained.

On January 15, the patient was improved. Paraphasia was still marked but there were moments in which she recognized her daughter, the hospital room and so on. She took fluids by mouth. She was quiet and drowsy and understood when she was spoken to. She spoke much but indistinctly, only once in a while she could be understood. On January 16, the hemiparesis on the right side was definitely diminished. She ate and drank, offered assistance when she got injections or when she was cleaned. Paraphasia and alexia were evident. She was asked, "Is that ice cream?" She smiled and answered, "I have entirely forgotten." On January 16 and January 17, the temperature rose to 102.4 rectally without corresponding increase of pulse rate (Fig. 2). Her fever was probably due to sulfonamides which were discontinued. There were 3,320,000 erythrocytes and

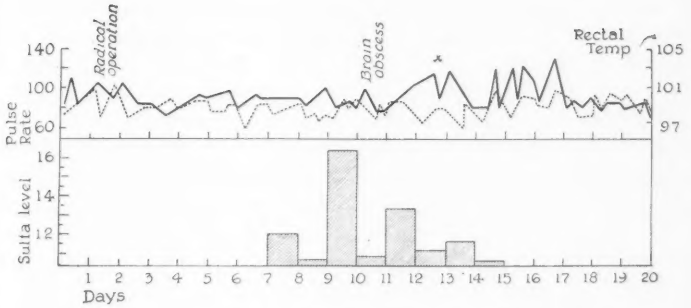

Fig. 2, Case 3.—Diagram indicating pulse rate, temperature and sulfa level. In *x* note the divergence between temperature and pulse rate.

8,100 leucocytes (juvenile forms 6%, polynuclear cells 46%, eosinophiles 6%, lymphocytes 40%, monocytes 2%) in the blood. Patient was alert despite her aphasia and deafness, she ate and drank well. There was a small brain herniation filling the epitympanum. On January 18 there were 3,750,000 erythrocytes and 6,500 leucocytes (juvenile forms 2%, polynuclear cells 35%, eosinophiles 2% and lymphocytes 61% in the blood. On January 20, there were 3,350,000 erythrocytes and 11,100 leucocytes (juvenile forms 4%, polynuclear cells 48%, eosinophiles 2%, lymphocytes 46%) in the blood. On January 24, the patient was well informed, also about her aphasia. The brain herniation was the size of a plum, was covered with granulations and did not show rhythmic pulsations. On January 27, there were 3,850,000 erythrocytes and 8,400 leucocytes (juvenile forms 5%, polynuclear cells 39%, eosinophiles 7%, lymphocytes 48%, monocytes 1%) in the blood. On January 28, all antibiotics were discontinued. On January 30, she was without fever for the last 10 days. The aphasia was marked, visions field could not be examined because of poor cooperation. On February 9 she was well but complained of periodical severe buzzing in the head which lasted for one-half to one hour. The brain herniation was small, soft and covered with granulations and connective tissue. Aphasia was evident. On March 18, 1947, she had gained weight. There was severe buzzing in the left ear. Amnestic aphasia was evident. There was a retroauricular fistula and a narrowing of the left external canal which contained a small amount of fetid pus. There was no paresis. On June 9, 1947, she spoke much better but still failed to use the correct names for colors. The left external canal was narrow; in the depth there was a membrane and some secretion which became fetid if she did not use hydrogen peroxide. There was a fistula in the posterior wall of the external canal leading into a channel which ran under the skin of the mastoid.

She was deaf on the left side and on the right side the hearing was markedly diminished. Nevertheless she refused to wear a hearing aid. On October 12, 1947, she felt well. There was a very small amount of secretion on the left side. Her speech was normal but it became twisted when she got tired. On August, 1948, she complained of periodical neuralgiform pain but she seldom had headaches. Her speech was good; occasionally she noticed difficulties in finding words. She had gained weight. The superior wall of the external canal was sagging and there was a slight purulent secretion from the attic.

Treatment: For one month the patient received heavy doses of penicillin, administered intramuscularly, intravenously and intrathecally. Five days following the operation sulfadiazine was added and maintained for one week.

In this case there was again a brain abscess on the left side which —in contrast to case 2—was caused by a chronic otitis associated with cholesteatoma. The exact duration of the abscess cannot be established as it is customary in instances of chronic otitis. It is possible that the initial phase of the abscess occurred about 3 weeks prior to the operation when the patient suddenly noticed earache, dizziness, headache and dullness of the sensorium. However, there is no proof because it is quite possible that she has had similar episodes—perhaps of minor degree—in the past which have passed unnoticed, inasmuch as the patient was apparently very careless with her ears.

The correct diagnosis was not made immediately since there was an acute, diffuse labyrinthitis which, at first, was considered to be purulent in character. When the acute symptoms of labyrinthitis had disappeared a radical mastoid operation was performed and both, a fistula of the labyrinth and an external pachymeningitis were discovered. After the operation the labyrinthitis subsided and the symptoms of a left-sided temporal lobe abscess became manifest, and 9 days following the radical mastoid operation, the temporal lobe abscess was drained. As in case 2 the abscess was in the latent phase.

It was not possible to use the original open method in this case because meninges and brain were firmly grown together as to render the preparation of the dura flaps impossible. For this reason a procedure was employed which, short of extirpation of the abscess, is probably the most radical procedure to be used in cerebral abscess. Two cannulas were introduced into the abscess cavity, one at the base and one at the convexity of the temporal lobe. The two cannulas met in the cavity. Between the two cannulas an excision of the tissue, down to the abscess cavity was made. The patient was cured without formation of a permanent brain herniation.

Interesting was the finding obtained by the puncture of the brain abscess. The fluid which escaped through the cannula resembled closely a cloudy spinal fluid and for this reason it was assumed that the cannula had entered the inferior horn of the ventricle. It took a few minutes until creamy pus was drained by the cannula. It is likely that the finding was caused by a sedimentation of the corpuscular elements of the exudate which sank to the floor of the abscess leaving behind a layer of cloudy fluid.

DISCUSSION

These cases were presented for the purpose of proving 1.(that in otogenous brain abscess the approach through the mastoid and the tympanic cavity is more logical than the approach through the vault

of the skull, 2.) that even radical surgery upon the brain can be performed through an infected field without taking the risk of a spreading infection of the brain and the meninges.

The writer prefers to use radical methods for the drainage of cerebral abscess because the less radical methods have not furnished satisfactory results in his hands. The writers experience is based on 35 otogenous temporal lobe abscesses. Four of these cases were discovered at autopsy. In the remaining 31 cases the following results were obtained:

	TOTAL	CURED	DEATH
Old method	25	7	18 (72%)
Open method	6	5	1 (17%)

The improvement of the results is actually not as conspicuous as shown by the statistics because the difference of the total figures is great. Nevertheless, it is a fair conclusion to state that the open method has caused an actual improvement of the treatment even if further experience may prove that the improvement is not as great as shown by the figures.

This, of course, does not indicate that the "open method" is the only method to achieve good results in cerebral abscesses of otorhinogenic origin. But it is a method which is based on sound surgical principles and which furnishes good results if a correct technic is used. The most serious technical error consists of tearing adhesions between meninges and brain. In fact, in the only fatal case of this series death was probably caused by tearing adhesions between brain and meninges. For this reason the writer has previously advanced the opinion that the open method can not be used if there are firm adhesions. Case 3 of this paper proves that this opinion is not quite correct and demonstrates the technic to be used in instances with firm adhesions.

However, the "open method" has a few drawbacks. Two of these drawbacks should be mentioned. The open method is apparently not successful in otogenous encephalitis phlegmonosa[4] (acute brain abscess).

The second drawback is the eventual formation of a brain herniation which may interfere with the after-care of the mastoid cavity. In this respect the following findings were obtained in 5 cases which were cured by means of the open method: In 2 instances there was a primary brain herniation viz., immediately after exposure of the

cerebral cortex the brain bulged into the bone cavity. In both instances there was a pale cyanosis of the cerebral cortex and in both instances the brain herniation was persistent although there was some reduction in size. In both instances the open method was used in a re-operation. This means that the abscess was drained by another surgeon, using another approach, prior to the employment of the open method. In one case—according to a written report—the abscess was apparently incised, the opening was dilated and paraffin-gauze was introduced into the cavity. In the other case the abscess was drained through drill holes in the temporal squama and the abscess cavity was filled with penicillin solution (case 1 of this paper). In both instances a severe brain edema, which in one case was associated with an edema of the scalp,developed immediately after operation and in instances the edema was promptly relieved by the open method. It is a matter of conjecture as to whether the herniation could have been avoided if the "open method" would have been employed primarily.

In the remaining 3 cases there were small primary herniations in 2 instances (cases 2 and 3) which spontaneously subsided. In the third case there was a secondary herniation viz., there was no herniation at operation. On the contrary, after drainage of the abscess had been established, the brain fell back leaving an empty space between brain and dura. During the convalescence a herniation of the brain developed which 10 weeks after the operation subsided only to a small extent. In other words, although this case had been operated on in the quiescent phase as cases 2 and 3 of this paper and there was no brain edema at the time of surgery, a brain herniation developed after the operation which occupied the entire cavity of the temporal bone and subsided very slowly and only to a small extent. Naturally the question arises as to the cause of these findings. The writer believes that the answer is the insufficient after-care in the last mentioned case. At the time of the operation, neither the modern sulfonamides nor the antibiotics were available. The after-care consisted simply of application of x-rays, ointment and gentle compression and this was apparently not sufficient to prevent a postoperative brain edema.

The experience gained from these cases, leads to the following conclusions: The open method applied to otogenous cerebral abscesses which are in the quiescent phase, and performed through an infected field, does not cause considerable brain herniation, either primary or secondary, provided that a proper after-care is instituted by employing hypertonic solutions, sulfonamides and antibiotics. Repeated spinal punctures to diminish brain pressure, are

not absolutely necessary. If, however, a cerebral abscess is associated with a marked brain edema, a primary and permanent herniation is to be expected if the open method is used. For this reason, the open method is not an ideal approach in the presence of brain edema. Yet it is an excellent procedure to relieve rapidly a brain edema, consequently, it should be used—despite its drawbacks— in instances in which the brain edema menaces the life of the patient. The risk which is involved in this procedure, is not great because the brain herniation soon is changed into connective tissue containing several cysts. This tissue is quite resistant against infections in the neighboring mastoid and tympanic cavities. In fact, reinfections of brain herniations are not frequent.

417 Mt. Prospect Ave.

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The Scientific Papers of the American Otological Society

LXXXV

ADDRESS BY THE PRESIDENT

PHILLIP E. MELTZER, M.D.

BOSTON, MASS.

It is a rare privilege to open the 83rd meeting of the American Otological Society and make you welcome at the sessions that will follow. Every effort has been expended to make the program an interesting one; it will be even more so if you will participate freely in the discussions.

Nineteen-fifty marks the beginning of the second half of the 20th Century and thus affords us the opportunity of reviewing what we have accomplished in the first half of the century. Many of you have been active throughout these fifty years and know what developments have taken place; I need not describe them to you. You have seen many enthusiasms born, flourish and die. Some of the things we have seen have been quite revolutionary and had quite far-reaching effects, but in time newer discoveries and developments made their bid for attention and superseded the previous theories and techniques.

Each era of this past half century of otologic history has had its prophets, philosophers, thinkers. Each of them knew the high excitement that stirs men when a new principle is established, and that excitement has always communicated itself to others. In the first decade, the emphasis was on the physiology of the labyrinth, an interest kept alive to the present day. The brilliant work of the otologist, Robert Barany, will never be forgotten. There have been many refinements of the Barany technique for investigating the function of the labyrinth, but his work remains basic. The stimulus his contributions gave to the otologists who followed him in the study of the labyrinth, its physiology and its pathology, and the development of effective therapy, established one of the milestones of the past half-century.

American Otological Society Meeting, San Francisco, May, 1950.

In the second decade, the advances that took place in the development of laboratory techniques in bacteriology and pathology considerably furthered the importance of otology as a special entity in the field of medical sciences.

The third decade witnessed the practical development of electronic science. The physicist and the engineer together made possible the audiometer; the otologist carried their contribution one step further, applying it to what was to become a new and extremely valuable technique in testing human hearing. The entrance into the picture of the physiologist and psychologist was marked by the development of the Wever-Bray phenomena, opening a new vista for psycho-acoustics. It was a natural step that the physiologist, whose function it was to determine by what means we hear, should participate in this new discovery. This society has listened to many interesting papers by the men who made these contributions. Now, twenty years after the discovery of the Wever-Bray phenomena, we are still discussing it. These contributions by the physicists—Fletcher Wegel, Steinberg, Bunch and Bekesy—to name only a few—plus the contributions of the psychologists Wever, Bray, Stevens and Culler, and the physiologists Davis and Galambos, as well as the anatomists Guild, Bast and Anson—not to forget the otologists like Gordon Wilson, Fowler Sr. and Lurie—are a perfect example of how different branches of science can cooperate in accomplishing a common goal. No better example could be found for the absolute necessity there exists today for the correlation and integration of scientific knowledge, training and effort.

There should not be omitted, in this decade of achievement, the contributions made in the study and treatment of facial nerve paralysis and palsy by Sir Charles Ballance and Arthur Duel. Happily, the need for this type of surgery by the otologist has been greatly diminished in ratio to the reduction in the incidence of mastoiditis.

In the fourth decade there appeared on the otological horizon a new clinical entity with a specific symptomatology and a specific surgical management; petrositis. Credit for this clinical achivement belongs to Ramadier, Eagleton, Kopetsky and Almour.

During this decade one of the great chemical developments touching on medical therapeutics was the discovery of the sulfonamides, a discovery that markedly reduced the incidence of, as well as the post-operative management, of otitic complications.

The second otologic contribution of revolutionary importance was the announcement of the Lempert one-stage fenestration operation. The surgical management of otosclerosis initiated by Holmgren and Sourdille, excited hardly any interest among otologists until, in 1938, Lempert announced his approach to the temporal bone by the endaural route which started a new trend for the exploration of the temporal bone. The last ten years have seen the perfection of the fenestration operation to a very encouraging degree.

The fifth decade has witnessed the great chemical engineering achievement that gave the world an abundance of antibiotics. This far reaching advance in therapy has changed our entire thinking in otology. For the first time we have an agent in the use of which we feel secure in combating the ravages of sepsis. It is encouraging that this decade has seen the surgical management of Meniere's Disease again back in the hands of the otologist.

This decade has witnessed, also, on a scale heretofore undreamed, a renewed interest in the plight of the deaf. In this respect the prominent names that come to mind are those of Newhart, Goldstein, Fowler Sr. and Berry. This interest was, of course, unquestionably hastened by the recent war, which suddenly plunged into the strange world of the deaf thousands of young men who had never known any physical handicap.

We are now witnessing the beginning of a new science; audiology. What part the otologist will play here is a question. Shall we be obliged to develop a new type of otologist for this special field—one trained as a psychologist, sociologist and educator as well as trained in the treatment of aural disease? This is going to be a very interesting development to watch. I have a feeling that the audiologist will simply be a contributor to otology and that his field will be one quite apart from that of the medical otologist as we who belong to the older school understand him to be. His relation to the otologist will be of a similar status as the physiologists and psychologist, but on a closer basis.

Thus we stand in mid-century, awaiting the venturesome philosopher to open new gates into the unknown. The contributions by the 20th Century otologist, limited as they may be compared to the contributions in general sciences, are nevertheless brilliant. They will not be forgotten in the chronicle of otologic history. We can be

proud of these confreres of ours who have so brilliantly contributed to the world's scientific knowledge.

I should like now to pass on to a quite different subject, one very dear to me, the training of the future otologist. I am very much concerned with the implications of the present status of the voluntary hospital staff as contrasted with a full-time hospital staff, implications that affect the young intern of tomorrow.

It would be absurd for me to venture an opinion on the merits of each system. I have had all my training under the so-called voluntary plan. In my own intern days, the discipline was rigid. Respect for authority, punctuality, regularity of attendance and integrity were the order of the day. The benefits one derived in return were immeasurable. The senior staff of surgeons and associate-surgeons earned their place in the hospital hierarchy by long years of regular attendance. Under their supervision, the junior staff found abundant opportunity to develop clinical judgment and surgical skill, and were trained to take over the responsibilities of the hospital.

As an illustration of the opportunity given the younger men for acquiring skill and training in the periods of 1920 to 1940, I have some figures here for you. There were 694 mastoid operations performed in 1929. In 1939 there were 442 operations on the mastoid, and in 1949 the operations were reduced to 139.

These operations were both simple and radical mastoidectomies. I should say here that our patient-load, that is, the total number of patients visiting the ear service, has remained fairly constant. It is particularly significant then that the number of operations has dropped from 694 to 139.

As I look at the situation, the problem of manning the outpatient department and teaching the interns, as well as supplying surgical opportunities to the voluntary visiting staff, who feel they are entitled to these opportunities, is a critical one. Whether this is a temporary situation or not, I cannot say. But from what I have heard about the circumstances in similar institutions elsewhere I doubt if the voluntary staff system in these hospitals will work out favorably, should it continue to function as it does now. Do we have the clinical material to give the kind of training, not to just four or six interns, but to as many as twenty or thirty men on the staff, that they must have?

We cannot avoid the economic element that enters into such considerations as these. Men who have spent a number of years in the armed services, for example, and who are now somewhere in the vicinity of 35 or 45 years of age, find themselves in a position where they feel they have to give first call to the demands of their private patients and leave what time is left for their obligations as members of this or that voluntary hospital staff. In a metropolitan center such as ours, where private beds are very hard to get for patients, men are often obliged to serve in two or three hospitals in order to have a chance at getting an occasional bed for their own private patients. They have to take whatever operating time is available, and this often interferes with their obligations elsewhere. Many of them find it necessary to take on paid positions in various hospitals or small clinics, to augment their earnings.

I understand what the problems are that the practicing doctor has to meet. But I also know how much indifference can jeopardize efficient hospital service. It is sometimes necessary to institute disciplinary measures that rarely prove satisfactory. Fortunately, there are always a few men on a staff who can be relied upon to do their jobs well. But the uncertainty of having available such men in sufficient numbers and the fact that the work-load sooner or later becomes unbearable undermines morale and creates a distinctly unfavorable attitude of mind and spirit. I have seen this sort of deterioration of morale take place, and I dare say that if we allow these conditions to continue we will not be able to maintain efficient public service or provide efficient teaching facilities for our undergraduates and interns. I believe that the voluntary system as it exists today will have to be modified and that the deficiency in staff numbers will have to be compensated for by the creation of a full-time hospital staff. The teaching of the undergraduate, the intern and a limited volunteer staff can be regulated and controlled with an efficiency that will benefit all. I do not advocate the dismissal of the volunteer staff. What I suggest is limiting this staff to men who are willing to make some sacrifice for the benefits of working with a full time staff in an institution where teaching is at a high level, where clinical discussions are organized and where an affiliation with a medical school is advantageous to their prestige and progress. These volunteers may have abilities and skills which exceed those of a full-time staff; the exchange of ideas and skills between the full-time staff and such men will be to their mutual advantage. The position of

the volunteer staff member in the community and in the profession would be on a par with their full-time associates.

There is another question, not irrelevant to this, that I should like to put to you. Should a large institution such as I envisage, one affiliated with a medical school, where teaching material is abundant, make itself available indiscriminately to applicants for internship of every degree of ability? What is the point in teaching a young man with only average aptitude the difficult procedures of modern otology? I recognize the impossibility of assessing with exactness the quality of work a young graduate will turn out in his internship or of determining at this early point in his career whether he will ever practice in a large enough medical center to exercise in full the training he receives. Certainly, if we do know in advance that such a man intends to practice in a small community offering limited opportunities or that he himself has limited capacities, we ought to organize ourselves so that we can give this type of graduate an internship in an institution that will give him the kind of training that will serve him best.

On the other hand, there should be available to the young men of more than average promise, institutions especially equipped for their training as otologic specialists. Medical graduates, on the basis of academic performance and previous training who have shown promise of exceptional ability should be invited to join the staff of the country's best hospitals for clinical training. A hospital that has earned itself a reputation for its teaching will have no problem in getting the kind of interns I have described. These hospitals should, as a matter of fact, make a deliberate effort to attract the best men by acquainting them with the opportunities they offer for postgraduate training. Competition among such hospitals for the best young men would be an excellent stimulus to the maintenance of high-quality teaching.

It was not so long ago that four cities in this country were dominant in preparing and teaching otolaryngologists. The younger graduates of these larger institutions have settled throughout the country and have raised the standard of otology to a great degree. With the development of the state universities and the university hospitals, the number of full-time staffs has increased. In recent years, the advantage of the full-time staff in the training of the younger interns and in service to the public has been proved time

and again. We who examine these graduates of state university medical schools for certification by the American Board of Otolaryngology have seen ample evidence of it.

The ideal institution, in my estimation, would be one which is a part of or affiliated with a medical school. The department of otology or otolaryngology would preferrably be autonomous. The chief of staff should have capable associates, men of experience in teaching and in surgical techniques. The residents should be chosen from among the interns who have completed their three year course and who are invited to remain for two more years. Residents should be given more than a marginal living salary. At the end of their residency, if they show unusual proficiency, they may be asked to join the permanent staff, or be at liberty to find positions or create opportunities elsewhere for themselves. With the background they would have acquired we can be sure there would be no difficulty in assuring their absorption in locations where they can exercise to the fullest the knowledge and skill their training has given them.

The size of a full time staff depends on the number of capable volunteers. This is not the place for me to discuss how such staffs can be financed, but many examples offer themselves of the successful financing of such a staff, and as far as I can see, it will not be a problem of any consequence.

This to me is the ideal type of service we can aim for and one that will assure the future of otology.

Before closing, I would like to make a few remarks about our membership. The by-laws governing qualifications for membership are just. I have no quarrel with them. My only suggestion would be in the direction of increasing the number of younger members. I have noted with pleasure how beneficial elections of young men have always proved in the past, beneficial not only to the new members but to ourselves. Young men have the vigor and the temerity to undertake projects that we older members sometimes shy at. The younger men are encouraged to do original work by election to a society such as ours. The older members gain even more in terms of new blood and new enthusiasm.

I would recommend going one step further and actively searching out young men of promise, wherever they may be, and invite them to speak before our scientific sessions and take part in local programs.

Let us reward them for the ability they have shown. In truth, it is we who will reap the greater reward. We can learn much in this respect from the practices of big business, which insures itself by selecting their future executives from among promising schoolboys, helping them acquire college or technical training and then placing them in their own laboratories, plants or offices for training in the field. This procedure has proved to be one of business' best investments. We could with great benefit to ourselves do precisely the same thing. Geniuses are only too rare, everywhere. Let us of the American Otological Society seek out the young men of promise, wherever they may be, and bring them into our fellowship.

20 CHARLESGATE WEST.

LXXXVI

MÉNIÈRE'S DISEASE: PRESENT CONCEPTS OF DIAGNOSIS AND MANAGEMENT

KENNETH M. DAY, M.D.

PITTSBURGH, PA.

It is unfortunate that Ménière's name is associated with a symptom complex and also with a disease entity which exhibits the same symptom complex or syndrome. The result has been a continued confusion in the minds of the medical public as to which is which. The term "Ménière's disease" has been applied for many years to cases exhibiting the symptom complex without evident cause or demonstrable pathology. As a result it has become a trash pile for a large group of cases with labyrinthine vertigo but without otitic suppuration or obvious intracranial disturbances. The demonstration by Hallpike and Cairns[11] in 1938 of the histopathologic changes in the labyrinth which occur in Ménière's disease served as a stimulus to otologists for a more intensive study of this condition, and has resulted in more contributions to medical literature on the subject during the past seven years than in the preceding half century. The histopathologic findings of an endolymphatic distention and a complete absence of inflammatory reaction have been confirmed by many. Because of the prevailing tendency to use the terms "Ménière's disease" and "Ménière's Syndrome" interchangeably, unsuccessful attempts have been made to change the name of the disease and to label it with a term describing the pathologic condition. Thus, the terms hydrops of the labyrinth, endolymphatic hydrops and labyrinthine dropsy have been used. However, after reading Williams's[17] review of the original papers of Prosper Ménière, one is forced to the conclusion that Ménière knew more about the disease which bears his name than many of the present day otolaryngologists and deserves the honor of having this disease entity named after him.

The etiology of Ménière's disease is as yet unknown. More and more evidence is accumulating to support the theory that an autonomic dysfunction or intrinsic allergy is the basic cause of this disorder which well might be called a vasomotor labyrinthitis. In an

occasional case a specific protein allergen may prove to be the causative factor. I have observed several cases in which the symptoms could be abolished on removal of offending allergens only to recur with their reintroduction. I shall present evidence that in Ménière's disease the cochlea is primarily affected and that the vestibular disturbance is a secondary or overflow reaction. This supports Williams's[18] theory that Ménière's disease is a form of allergy involving the capillary loops of the stria vascularis and that the endolymphatic distention is a localized extracellular edema produced by the spastic-atonic state of these capillaries. I cannot agree with Atkinson's[2] theory that specific vitamin deficiencies and nutritional disturbances are the causative factors of Ménière's disease. If his theory were true, why is it that bilateral involvement is so uncommon? It seems worthy of comment that several hundreds of cases of unilateral Ménière's disease have been operated upon successfully without subsequent appearance of symptoms from the uninvolved ear in spite of the fact that no vitamin therapy or dietary regulation has been prescribed for them. Atkinson apparently groups all cases with Ménière's syndrome together and fails to differentiate the cases of true Ménière's disease from those without evidence of endolymphatic distention. Ménière's syndrome of deafness, tinnitus and vertigo is not uncommonly present in cardiovascular disorders and frequently is associated with cerebral arteriosclerosis. It also occurs in cases of so-called toxic labyrinthitis. The deafness is usually a perceptive deafness of the upper scale, and the tinnitus is high pitched. I do not mean to imply that vitamin and nutritional deficiencies may not cause vertigo and deafness, but I do not believe that they are primary factors in causing the endolymphatic distention of true Ménière's disease. I have been able to confirm the diagnosis of Ménière's disease in not more than one-half of the cases referred to me with such a diagnosis during the past ten years.

The diagnosis of unilateral Ménière's disease is rarely difficult if one keeps in mind the labyrinth changes present in this condition. The symptoms and signs fit accurately the picture one would expect to see if there were a fluid pressure exerted on the endorgans of the labyrinth. There is a diminution and distortion of response to stimulation. The deafness is usually a uniform loss of hearing throughout the scale with decreased bone conduction, diplacusis and severe recruitment. Mygind[13] calls this an inner ear deafness of the conductive type. The degree of deafness often fluctuates indicating a

variation of the endolymphatic pressure. Associated with the deafness is a low-pitched roaring or humming tinnitus which is almost pathognomonic of Ménière's disease. This tinnitus should not be confused with low-pitched throbbing or pulsating vascular tinnitus. The vertiginous attacks are due to discoordination of the sensory responses of the two opposing vestibular mechanisms. This would be analogous to the reaction of a bimotored plane with one motor skipping. The first symptom which comes to mind at the mention of Ménière's syndrome is vertigo, but vertigo is often a late symptom of Ménière's disease just as a stiff neck is a late sign of meningitis. Vertigo and the frequently concomitant symptoms of nausea and vomiting are so incapacitating that they overshadow and submerge the more important cochlear symptoms.

A recent review of the case histories of 220 patients seen during the past seven years reveals some interesting information. This series includes 175 cases of typical Ménière's disease and 45 cases diagnosed as Ménière's disease but without vertigo.

TABLE I

Deafness but no vertigo	45
(a) Onset of deafness before vertigo	83
(b) Deafness and vertigo noticed together	83
(c) Vertigo noticed before deafness	5
Not noted (poor histories)	4
Total	220

(a) The average elapsed time between onset of deafness and onset of vertigo was 3½ years.

(b) Deafness may have been present in some of these cases but was not noticed prior to onset of vertigo.

(c) Two of these cases did not complain of deafness at time of examination, but audiograms revealed an average loss of 30 and 50 decibels respectively. Three cases had vertigo attacks for from one to three years before deafness was noticed.

A study of Table I seems to justify the following conclusions: (1) Ménière's disease is primarily a cochlear disturbance. (2) When

the endolymphatic distention is limited to the ductus cochlearis there will be no vestibular symptoms. (3) It is probable that distention of the saceule and utricle do not occur as a primary condition but only following an overflow of endolymph from the distended cochlear duct. This may occur with the initial disturbance, or it may be delayed for months or for years.

Of the 220 cases being reviewed, 27 showed bilateral involvement, and of these, 23 gave a history of simultaneous involvement of the two labyrinths. In the remaining four cases, the elapsed time before involvement of the second ear was one year, three years, seven years and 29 years. From these figures, one may assume that late or secondary involvement of an unaffected ear is rare.

A positive history of allergy was obtained in 45 of the 175 cases of typical Ménière's disease, and in 23 of the 45 cases without vertigo.

TABLE II

MÉNIÈRE'S DISEASE WITH VERTIGO—175 CASES

Tinnitus—constant low pitched (100 to 400 cycles) 157. Low pitched (only with attacks) 6. High pitched 5. Absent 7.

Diplacusis—Noted 128. Not noted 24. Absent 23 (bilateral 15).

Subjective Recruitment—Noted 90. Not noted 85.

TABLE III

MÉNIÈRE'S DISEASE WITHOUT VERTIGO—45 CASES

	under 1 year	1-3 years	3-5 years	over 5 years
Duration	21	10	9	5
	low pitch	high pitch	absent	not noted
Tinnitus	40	2	1	2

Diplacusis noted in 29 cases. (Noted in only 2 of 6 bilaterals):

Fluctuation of hearing loss noted in 22.

Subjective recruitment noted in 14.

The tinnitus was usually described as a roar, hum or seashell noise if pitched around 100 cycles. When described as a hiss or escaping steam, the pitch was usually about 400 cycles. The pitch of the tinnitus should be tested as some patients will confuse pitch and intensity.

Diplacusis is a very important diagnostic sign or symptom and should be tested. The absence of diplacusis in a number of cases in Tables 2 and 3 was because of involvement of both ears or because the deafness was too profound for diplacusis to be detected.

Variation of the degree of deafness is another important symptom in both groups and of special value in establishing the diagnosis of Ménière's disease without vertigo. It was noted in the histories of 22 of the 45 cases in this group.

Subjective recruitment or hypersensitivity to loud sounds is a symptom frequently overlooked in history taking, yet it was noted in 90 or over one-half of the cases in Table 2. It is really a symptom of over recruitment meaning that outside noises sound louder in the deafened ear than in the unaffected ear. Fowler's[9] recruitment phenomenon has received too little attention from otologists. It is a simple matter to test for recruitment if one has two headphones with an audiometer. A recent contribution by Dix, Hallpike and Hood[8] apparently proves that recruitment does not occur when deafness is due to a lesion of the 8th nerve but only when the cochlea itself is affected. They performed loudness balance tests on a number of proven cases of 8th nerve lesions and found recruitment to be notably absent unless there were secondary vascular changes affecting the cochlea. On the other hand, in cases of Ménière's disease, complete recruitment was the rule and over recruitment was common. The results of their tests were illustrated by ladder diagrams. It is now the rule on my service to chart laddergrams on all cases of unilateral nerve or perceptive deafness (Fig. 1).

In brief, the main cochlear symptoms of Ménière's disease are a fluctuating degree of deafness involving the entire scale, decreased bone conduction, low-pitched tinnitus, diplacusis and hypersensitivity to loud sounds.

The attacks of vertigo are, of course, typical of peripheral, labyrinth disturbances. They are usually explosive or paroxysmal and occur with or without any warning aura. The secondary symp-

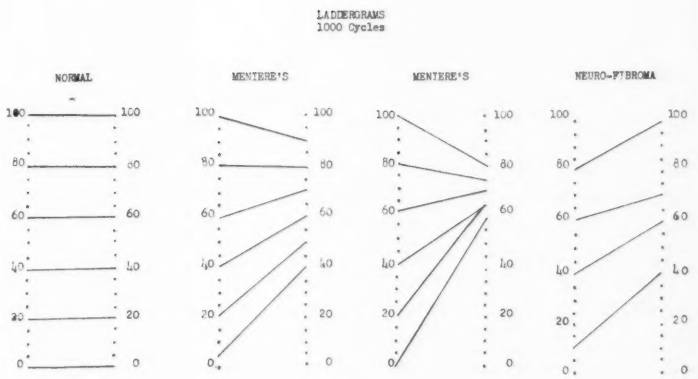

Fig. 1.

toms of nausea and vomiting are simply indicative of the severity of the attacks of vertigo. The attacks vary in frequency, duration and severity. They may last from a few seconds to several hours. Many patients can foretell the approach of an attack by a change in the pitch or intensity of the tinnitus. Sometimes the deafness becomes more profound just prior to an attack, though occasionally the hearing will improve. During the intervals between acute attacks, many patients have a constant feeling of unsteadiness and a vague sense of pressure in the head. Remissions may occur with or without treatment and last for months or years. During periods of remission, however, the deafness, tinnitus and diplacusis continue to be present. Caloric tests uniformly elicit diminished responses from the affected labyrinth. The responses from the two labyrinths may be equal or the affected labyrinth give the lesser response. A hyperactive labyrinth should never be found in a case of true Ménière's disease.

The only difficult diagnostic problem is between Ménière's disease and toxic labyrinthitis with perceptive or nerve deafness, tinnitus and varying degrees of vertigo. In the latter bilateral involvement is the rule rather than the exception and vitamin and nutritional deficiencies are common. It is sometimes difficult to distinguish from true Ménière's disease. However, the hearing loss frequently affects the high tones more than the low tones, tinnitus

is usually high pitched and there is little, if any, fluctuation of the degree of deafness. Diplacusis is slight, if present at all, and recruitment, while present, is not severe. Vertigo is often continuous and paroxysmal attacks rarely occur. Caloric tests are apt to show hyperactivity. Some of these cases with toxic or nutritional disturbances have vertigo without cochlear symptoms and vice versa.

I have nothing new to offer on the conservative treatment of Ménière's disease except to stress the importance of early treatment. If possible, treatment should be instituted before the onset of attacks of vertigo. When both cochlea and vestibule are involved in the endolymphatic distention and the process has become full-blown, the most that can be expected from conservative treatment is palliation. Once the cochlear duct has become distended to many times its normal size, it should be obvious that the duct cannot return to normal. I agree with Altman[1] and Williams[19] that in such cases conservative treatment may smother and control the acute attacks but cannot be expected to effect a cure. I cannot give accurate figures on the results of treatment in my series of cases since much of the treatment was not done under my supervision. I can say that restoration of practical hearing and relief from tinnitus occurred in approximately one-half of the cases of Ménière's disease without vertigo when treatment was begun during the first year following the onset of symptoms. The longer the symptoms have persisted, the less effective is treatment. Even if early treatment does not succeed in restoring the hearing loss, it may be effective in checking or retarding the onset of attacks of vertigo. Treatment should consist not only of dietary regulation and the use of such vasodilators as histamine and nicotinic acid, but the patient also should be studied as a whole and a thorough physical examination made with special reference to metabolic and endocrine disturbances. Any irregularities should be corrected. Psychotherapy is sometimes helpful, especially if the onset of attacks can be traced to emotional upsets or mental and nervous fatigue. It is very difficult to evaluate the course of these cases over a long period of time since many of the cases, which we consider to be controlled by treatment or to be in a period of remission, disappear from sight. Later, to our chagrin, we learn that they have had a recurrence of attacks and have consulted other doctors.

Operative intervention is indicated for any case of unilateral Ménière's disease in which conservative treatment fails to control the

attacks of vertigo. Where loss of time from work because of attacks creates an economic hardship or, when the patient is so situated that continued treatment and observation cannot be carried out, then surgery is the treatment of choice. Surgery is definitely indicated when the attacks of vertigo occur without warning and are so severe that the patient falls or if he be engaged in an occupation where a fall might endanger life. Surgery is the preferable form of treatment when the psychosomatic disturbance is severe. The fear of attacks is a major symptom in many of these cases and may persist for a long time after actual attacks have ceased. Delayed rehabilitation following surgery is frequently due to this factor alone. I do not consider advancing age to be a contraindication to surgery. Some of the most grateful patients upon whom I have operated were in the late sixties and seventies. If both ears are affected, I do not believe that surgery of the labyrinth is justified. I operated on one labyrinth in two such cases several years ago. One case secured partial relief following operation plus histamine therapy which controlled acute attacks, but the hearing of the unoperated ear rapidly diminished, and the patient was nearly deaf at the time of his death from cardiovascular disease two years later. The other case was not helped by operation. With bilateral involvement, if other conservative treatment prove ineffective, consideration should be given to streptomycin therapy.

There is still a difference of opinion as to the choice of operative procedures for Ménière's disease. Portmann's[15] endolymphatic sac operation has had some success, but the frequency of recurrence of symptoms following this procedure definitely limits its value. Partial or complete section of the 8th cranial nerve is still practiced by some of the neurosurgeons. It is a major undertaking, entailing the opening of the arachnoid spaces. The mortality rate is not insignificant and damage to adjacent nerves and to the cerebellum itself is not uncommon. If partial section be performed with preservation of hearing, there is likely to be a preservation of some vertigo. I should like to see a report on the practical value for speech with discrimination scores of the hearing which has been preserved by partial nerve section. In my own series of cases when I attempted differential electrocoagulation of the vestibular mechanism without damage to the cochlea, I was none too happy about some of the results when hearing was preserved and I found that the retained hearing was more often a detriment than an asset. At the present time the type of

operation in general favor seems to be a labyrinthotomy by way of the mastoid approach with destruction of the vestibular mechanism through an opening in the horizontal semicircular canal. This can be done by the Cawthorne[3] method of removing the membranous horizontal canal, by my method of electrocoagulation of the vestibular contents or by the insertion of a barbed dental broach into the vestibule to destroy its contents. Consistently successful results have been reported from these labyrinth operations so far as the attacks of vertigo are concerned. Preservation of hearing has been an uncommon occurrence. To date I have heard of only one fatality due to a cerebellar abscess following this type of operation. There have been reports of several instances of facial nerve injury resulting from the use of the coagulating needle electrode, but this should not occur with proper surgical technique. For simplicity, safety and consistently good results, operation on the labyrinth seems to be by far the preferable procedure. The possibility of subsequent involvement of the unaffected ear is used as an apparently strong argument against labyrinth surgery for this condition. To date I have not seen this occur. Williams[19] makes a similar observation; Sullivan[16] reports one case out of 72; Furstenberg,[10] none out of 30; Cawthorne[4] reports several cases with subsequent involvement of the unoperated ear and comments on the predominance of the cochlear symptoms. The fact that subsequent involvement of the second ear was noted in only four of 220 cases mentioned earlier in this report tends to confirm my opinion that its occurrence is so rare as to be negligible.

Recently, Passe and Seymour[14] have reported favorable results in a small series of cases in which an entirely different operative approach was attempted. Their procedure consists of the removal of the stellate ganglion on the affected side, division of T 1 and T 2 and stripping or section of the vertebral artery. Their selection of cases was not limited to cases of true Ménière's disease and their reported results need confirmation from other sources. Nevertheless, this is by no means an illogical approach in that the purpose of the operation is to interrupt the vasoconstrictor fibres to the internal auditory artery. In one of my cases with bilateral involvement, this operative procedure was carried out on the right side with section of the vertebral artery. There has been a further loss of hearing in the right ear during the ensuing three months, and vertigo attacks and roaring tinnitus have continued. The patient is now receiving injections of novocaine into the left stellate ganglion. There has

been no noticeable change in his condition following two such injections. Lima[12] reports on angiographic studies following resection of the superior cervical sympathetic chain. He found that such a procedure had little effect on the cerebral circulation and that even bilateral sympathectomy did not cause vasodilatation comparable to that seen in the extremities.

Up to the present time, I have operated upon 66 cases of Ménière's disease and a report of the results may be of interest. I have routinely used the same operative procedure which I have previously described;[5-6] a mastoid antrotomy and labyrinthotomy with electrocoagulation of the vestibule. The only postoperative complication in my series has been the development in two cases of a haematoma which required simple evacuation. There have been no postoperative infections to date, and no case has developed a facial weakness. For several years I attempted to preserve hearing in all my cases and was successful in 15 of the first 53 cases including eight of the last 15 attempts. This group of 15 cases might be considered to be comparable to a group of cases with retained hearing following differential section of the vestibular nerve. The hearing which was preserved in most of my cases has gradually deteriorated so that at the present time I can claim only four cases with hearing maintained at the preoperative level or better and without severe distortion and tinnitus for a period of from 18 months to five years following operation. The hearing curve of one of these cases is illustrated as an incongruity (Fig. 2), though I doubt very much if it will be like this five years from now. Seven patients have complained bitterly of roaring tinnitus and distortion, and of these, four have had a recurrence of attacks of vertigo apparently arising from the posterior semicircular canal. Revisions were done on these four cases. On revision the horizontal semicircular canal was found to be obliterated by bone in all cases, and a mound of bone bulged forward into the vestibule. In two of these cases, the ampullated end of the superior canal was also opened and found to be occluded by bone. Postoperative vertigo in these cases was found to be similar to that following the primary operation. Surprisingly, one case still showed hearing preserved at the preoperative level following revision, and a second revision was later done to destroy the hearing and eliminate the roaring tinnitus. A revision was also done on another case, at the patient's insistence, because of roaring tinnitus, although he had been free of vertigo for over a year following the primary operation.

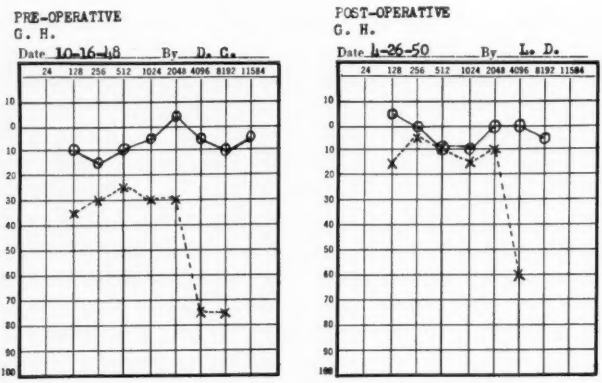

Fig. 2.

Because of these poor results when hearing was preserved and, because of the added possibility of incomplete destruction of the vestibular mechanism, I no longer attempt or advocate differential electrocoagulation. By the same token I question the value of differential nerve section except in the rare case when hearing loss and distortion are very slight. The preservation of hearing in some of my cases was probably due to the fact that the needle was not passed deeply enough to destroy the saccule. This was proved by temporal bone sections in one case reported by Lindsay and Day.[7] I now use a curved needle with which I can also reach the area of the ampullated end of the posterior semicircular canal. To insure complete destruction of both vestibular and cochlear function, the needle electrode should be passed deep into the vestibule and two or three applications of current made with the needle in this position.

Of the 51 cases of my series in which hearing was destroyed by the initial operation, all but the two bilateral cases have been free from acute vertigo attacks and have been rehabilitated.

I recently mailed questionnaires to all the operated patients whom I could trace and received 51 replies out of a possible 57. These questionnaires were used in order to get the subjective reactions of the various patients to the amount of benefit attained from operation.

QUESTIONNAIRE—51 REPLIES

Any dizzy attacks since operation			No 45	Yes 6	
Any hearing in operated ear			No 40	Yes 11	
Hearing in unoperated ear	Less 3		Same 36	Better 12	
Any head noises			No 12	Yes 39	
Worse than before operation	4		Less 35		
High pitched	29				
Low pitched	10				
Any difficulty with balance			No 30	Yes 21	
Any difficulty with balance during			day 6	Night 21	
When did you return to work or resume your household duties.					
10 days to two weeks	9	3 weeks	8	four weeks	11
5-8 weeks	11	3 months	6	over 3 months	3
No answer	3				

Of the six patients reporting yes to dizzy attacks, one was a bilateral case, two suffered attacks of cerebral angiospasm, two said they had giddiness and one was probably due to incomplete destruction of the vestibular mechanism.

Two other questions were asked in the questionnaire. Were you benefited by the operation? Yes 50 No 1.

Under similar conditions would you undergo the operation again? Yes 50. No 1. The lone dissenter for these two questions was a bilateral case.

CONCLUSIONS

Ménière's disease is probably caused by an autonomic dysfunction or intrinsic allergy affecting the capillary loops of the stria vascularis.

This disease primarily affects the cochlea. Vestibular disturbances are due to secondary or late involvement of the vestibular mechanism.

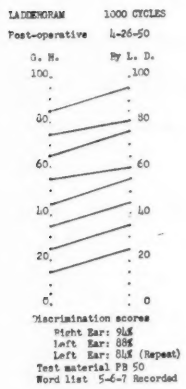

Fig. 3.

The diagnosis of Ménière's disease can and should be made from the cochlear symptoms alone.

Conservative treatment should be instituted before the vestibular mechanism becomes involved. If this be done, there is a good prognosis for restoration of hearing loss, relief from tinnitus and the prevention of attacks of vertigo.

When both cochlea and vestibule are involved in the endolymphatic distention, conservative treatment may be effective in controlling attacks of vertigo but will not effect a cure.

Destructive labyrinthotomy and 8th nerve section are equally effective in eliminating the acute attacks of vertigo caused by Ménière's disease. The former procedure is simpler, safer and requires a shorter period of hospitalization and convalescence.

Differential destruction of the vestibular mechanism with preservation of cochlear function has been successful in a number of cases but has not added to the well being of the patients. Cochlear distention has continued with associated distressing symptoms, and vertigo was not completely abolished in all cases.

These cases are considered to be comparable to cases which have undergone differential section of the 8th nerve.

The possibility of subsequent involvement of the unaffected ear by the disease is considered to be so rare as to be negligible.

At the present time destructive labyrinthotomy appears to be the operation of choice for unilateral Ménière's disease.

121 UNIVERSITY PLACE.

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LXXXVII

SUDDEN DEAFNESS

EDMUND PRINCE FOWLER, M.D.

NEW YORK, N. Y.

Sudden deafness may be caused by lesions of either the neural or the mechanical sound conduction mechanisms of the ear. It is an alarming experience whether it be moderate, severe, or total, and a shattering experience if it involves both ears in marked degree. Deafness is never total, and seldom very profound when due to sound impedance lesions alone, and such lesions are rarely sudden.

Just what does the term "sudden deafness" imply? How sudden is "sudden deafness?" To the patient it involves the realization that seemingly without warning, he no longer hears as well as he thought he was hearing, in the immediate or recent past. Sudden deafness may develop within a moment or so, within a few minutes, or after several hours. Many days, or even months may pass before the patient becomes aware of it. Within any of these time intervals the patient may call it sudden. Some people have been known to be totally deaf in one ear for years without being aware of it until they suddenly noticed that when one ear (the good ear) was buried in a pillow they could not hear a speaker standing a short distance away from them. This may have been sudden deafness earlier in life, but it is under the circumstances only sudden awareness or realization of deafness, not sudden deafness as of the time it was noted.

Sudden partial deafness may be, and often is, reversible when it is caused by impedance lesions and also when it it occurs with Ménièriform attacks. It is not reversible when it follows suppurative labyrinthitis, or degeneration of the auditory neural elements from any cause; it is then always permanent.

The incidence of sudden deafness of unknown origin is generally thought to be low. I have found it to be greater than for sudden deafness of known origin.

The etiology of sudden deafness has been hazy, mainly because this type of deafness is seldom associated with events or diseases which

kill the patient and, permit of autopsy following the calamity is not soon enough to correlate the clinical and autopsy findings. Moreover the histopathology in the few cases examined is usually of only partial help because it simulates that frequently found in deafened persons with various otological histories.

Herein we shall be concerned with the permanent sudden deafness (usually total in one ear and frequently total in both) which mysteriously occurs out of a clear sky without hitherto discernable cause. It often occurs in people who are apparently in good health. Heretofore all the otologist has definitely been able to say about such sudden deafness is that it is a "neural deafness." Generally he carelessly calls it a "nerve deafness" and foregoes an etiological diagnosis. When pressed it is customary to ascribe the etiology to a toxic destruction of the neural elements, or to acoustic trauma, or to a vascular accidnt, or to a virus disease, to drugs (quinine, mercury, barbituates, etc.) or to a transfusion Rh factor incompatibility. Sudden deafness is in the adult usually accompanied by tinnitus and vertigo at the onset. Kobrak[1] believes that given a sudden deafness with dizziness, hemorrhage should always be suspected even if there are no signs of hemorrhagic diathesis.

The term "vascular accident" implies a hemorrhage from or a thrombus or embolus in or affecting the ear vessels. Without convincing evidence it is unscientific to use this term definitely, and up to now there has been little direct evidence that this hypothesis is correct.

Thrombosis or embolism in the internal auditory artery or one of its branches presupposes either a vascular lesion, for example, arteriosclerosis or endarteritis obliterans as the cause of the thrombosis, or endocarditis, or some disease which could cause embolism. None of these disease entities have been proved to be the etiological factor in this type of sudden deafness, although one or more of them may have been present in a few incidences. Moreover this type of sudden deafness is frequently found in young and early middle aged people without vascular disease.

Many acute bacterial and virus diseases are followed by sudden deafness. Meningitis, scarlatina, influenza, mumps, rubella and typhus are well known examples. Frequently there is little direct evidence to definitely etiologically connect the pathology of disease itself with the deafness.

TABLE 1.—SUDDEN DEAFNESS—26 PATIENTS

Sex: M. 19 F. 7	Family History of Deafness	16
	No Deafness	10
Time of Examination:	Soon after onset	13
	Late	13
Age at Onset:	Men	Women
Under 40	13	6
Over 40	6	1

TABLE 2.—SUDDEN DEAFNESS—26 PATIENTS

	POS.	NEG.
Emotional episodes before onset	23	3
Fractured skull	1	
Mumps relapse	1	
Questionable positive Wassermann	1	

TABLE 3.—SUDDEN DEAFNESS

Severe deafness in both ears in	1
Total deafness in both ears in	5
Total deafness only in right ear	15
Total deafness only in left ear	5
	26

I have tabulated the findings in 26 cases of sudden deafness, noting everything I could think of which might have had some bearing on the etiology. Unfortunately most of the data seem unilluminating. I did not know exactly what to look for. Instead of reporting each case in detail I shall present a summary of the few findings seemingly pertinent to our discussion. Also I shall refer to sixteen cases recently reported by Lindsay,[2] and eighteen reported last year by Rasmussen,[3] thus furnishing in all sixty cases for discussion. Many more could be cited, but these will suffice for the present purpose.

Of my twenty-six cases Tables 1 to 5 set forth these findings: About 77% were under 40 years of age (Table 1).

With three exceptions there was a history of emotional episodes sometime before or immediately preceding the sudden deafness (Table 2). The patient rarely blamed the emotional condition for his deafness. One patient fractured his skull but was too drunk to know whether the liquor or the fracture immediately preceded the deafness. In one patient the deafness followed a relapse of mumps. One had a prior plus Wassermann. Otherwise with the exceptions noted, and the emotional factors, the routine neurological examinatoins were negative.

There was a binaural total of profound deafness in both ears in six and a monaural total deafness in twenty (Table 3).

Tinnitus was present in twenty and absent in six (Table 4).

Of the 30 totally deaf ears tinnitus was present in 20, absent in 10. Of the 11 partially deaf ears tinnitus was present in 7, absent in 4. Of the 11 near normal ears tinnitus was present in 2, absent in 9. Of the total of 52 deaf ears tinnitus was present in 29, absent in 23.

In 14 patients tinnitus was present in the affected ear just preceding the deafness. Six patients were uncertain about tinnitus just preceding the deafness. In six patients there was no remembered tinnitus in either ear at any time. The static labyrinths showed a reduced or no response on one or both sides in 21 patients (Table 5).

There was a history of vertigo before, during, or following the suddent deafness in 21 (accompanied by nausea or vomiting in 10).

TABLE 4.—SUDDEN DEAFNESS

Tinnitus present only in deaf ear	5
Tinnitus present only non-deaf ear	5
Tinnitus present in both deafened ears	10
Tinnitus absent in both deafened ears	6
	26

TABLE 5.—STATIC LABYRINTHS

No response either side	5
No response one side Lowered on other	4
Reduced response on one side	9
Reduced response on both sides	3
Increased response on both sides	1
Normal both sides	4
	26

TABLE 6.—STATIC LABYRINTHS

History of vertigo before, during or following the sudden deafness in	21
No vertigo	5

(Nausea and vomiting were present in 50%)

On January 16th, 1950, Lindsay and Zuidema reported sixteen cases of inner ear deafness of sudden onset.[2] Three were associated with ear complications and a systemic disease, one with epidemic parotitis, and twelve with unilateral sudden and permanent inner ear deafness, and varying degrees of vestibular disturbance of uncertain etiology. Two cases followed an acute upper respiratory infection.

Three other cases were reported by Lindsay with a syndrome of bilateral neural lesions, or inner ear dysfunctions, associated with an inflammatory ocular disturbance. Two of these had profound loss of auditory and vestibular function, and one a less severe disturbance followed by partial recovery of function. Lindsay assumes that this syndrome is toxic, and is associated with inflammatory processes of unknown etiology.

It was suggested that in the twelve cases with unknown etiology the clinical pictures strongly indicated a vascular accident in the labyrinth, or a toxic neuritis as the probable explanation.

Of Rasmussen's 18 cases of sudden deafness, (the total number seen in the last 15 years) 15 were women, 3 were men, ages 19 to 40. The lapse of time between the onset of deafness and the examination varied from 1 to 19 days with the exception of four cases seen after a greater lapse of time. All were unilateral and about equally distributed between the right and left sides. In three there had been a prior deafness in the opposite ear, and in three patients the acoustic apparatus alone was affected, while in 15 the vestibular labyrinth also was involved, with total deafness in eleven.

Vertigo more or less violent occurred immediately or a little after the attack, and was usually attended by nausea and vomiting. In a few cases the vertigo preceded the deafness.

The neurological examinations and Wassermann tests that were made were negative, as were the x-rays of the *porus acusticus internus*. The blood pressures were normal. Three of Rasmussen's patients had arteriosclerosis.

In all of these 60 cases of sudden deafness, there appears to have been a neuritis, or degeneration of the eighth nerve of unknown origin. It is probable that the degeneration was caused primarily by an anoxia, ischemia, and altered metabolism.

Vascular spasm in the inner ear cannot be diagnosed per se, but if it can be demonstrated somewhere else in the body it is probable that it also may have been present in other parts of the body; in parts of the neural apparatus of hearing. If sludging of the blood is observed it increases this probability because sludging of the blood is closely associated with vascular spasm, and no matter when sludge forms it is eventually carried by the blood to all areas and to all tissues. Food and other poisons also may cause neuritis of the eighth nerve, but also they are associated with sludging of the blood.

All five of my patients examined for sludging showed it distinctly. I believe all of the others would have revealed it had they been examined early. It is probable that the neural irritations and degenerations causing the sudden deafness, tinnitus and vertigo, under discussion were accompanied by, and at least in part caused by, the sludging phenomena, and the emotional episodes remembered or forgotten while awake or asleep. Such episodes are known to affect the neurovascular mechanisms through autonomic nervous system response; vascular contraction, sludging and eventually congestion, edema, anoxia and leakage from capillaries, and local metabolic deficiencies. All or any of these factors may be and frequently are the cause of sudden deafness.[4]

Two of my patients gave a history of liver disease, and 18 suffered from psychosomatic disorders. These latter included psychic shock, three, gastritis, ulcers or ulcerative colitis, two, rheumatism, four, autonomic instability, eight, and severe allergy, one.

Sudden deafness may occur in the presence of a chronic neural or impedance deafness. Just before I left New York for this meeting a patient of mine, a young man of 32, with a 30 db impedance loss in hearing, suddenly lost his father. Severe emotional episodes followed with a profound neural deafness which lasted four days. The blood was markedly sludging, and the circulation in the arterioles and venules of the eyes was definitely slowed in some areas.

In conclusion, therefore, we are proposing that the conjunctival vessels be searched for blood sludge to furnish further factual evidence that sudden deafness is due to changes in vascular supply. Previously this has been entirely inferred from the fact that spontaneous small hemorrhages and thrombi have been observed elsewhere in the body, notably in the retina producing sudden blind spots without known cause. It is hard to envision anything except a

vascular phenomenon of this type that could cause sudden deafness and the usual concommitent tinnitus and vertigo. The clumping of the blood called by Knisely "blood sludge" occurs so often and is such a logical explanation of such symptoms that we present it and its concommitent phenomena as a most probable etiological factor.

Blood sludge occurs in a large variety of conditions and can even be produced by certain hormones; adrenalin, estrogens and emotional upsets. It only occasionally causes symptoms that have been clearly recognizable in any organ. It is therefore probable that sludging only causes deafness when there is some hereditary anatomical or previous disease or injury which favors sludge sticking in the small terminal vessels of the cochlea. As we have before suggested, a transient sludge could well account for Meniereform attacks, and transient tinnitus, vertigo and deafness. A sludge which remains a sufficient length of time we believe accounts for the sudden deafness which is the subject of this paper.

Blood sludge as mentioned before can be produced with adrenalin, and accompanies estrogenic disorders. This proves that emotional upsets can be responsible for it and suggests that sudden deafness can be precipitated by emotional upsets. It has been shown that sludge can be dispersed by novocaine intravenously and by other measures. It is suggested, therefore, that in acute cases of sudden deafness with blood sludging, anti-sludging therapy be used; five hundred cc of one-tenth percent novocaine may be used intravenously in an initial effort to dislodge the sludge. Such therapy also has occasionally been successful in long-standing tinnitus. If our experience and our theories are correct it may be possible in early cases to relieve sudden deafness.

140 East 54th Street.

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LXXXVIII

AN ANALYTICAL SURVEY OF THE EVOLUTIONARY DEVELOPMENT OF THE FENESTRATION OPERATION

JULIUS LEMPERT, M.D.

NEW YORK, N. Y.

In the December 1937 issue of the Bulletin of the New York Academy of Medicine, Sourdille[1] who made a great contribution to the surgery for otosclerosis by developing a three stage surgical technic for exteriorizing and sealing the newly created fenestra within the external semicircular canal, stated as follows: "In a great number of cases, unfortunately, the success is ephemeral: Four, six, or ten weeks later, one sees the aerial hearing diminish, the Rinne becomes negative and the Weber indifferent. At the same time, the air pressure in the meatus can attain 40 and even 60 cm of water without determining nystagmus, nor a sensation of vertigo. This is due to the fact that the labyrinthine fistula closes, due to the reconstitution of a rigid bony layer, which rarely attains the thickness of the primitive bony wall of the canal, and more often does not exceed a few tenths of a millimeter. It suffices, in a complementary operation, to extract this bony film, to see the hearing gain of the first operation return, and sometimes be even greatly increased. This time the result will be lasting, the regenerating process of the bone becoming gradually exhausted. In many cases, however, I had to open the labyrinth three times."

"We must still work to solve this problem of osteogenesis, which will diminish not only the unpleasantness of reopening the labyrinth, but one of the causes of grave failure; the injury of the membranous canal."

Holmgren,[2] who pioneered in this surgery for many years and studied how to create a safe technic for making the fenestra without injury to the membranous labyrinth, and also attempted experiment-

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From the Lempert Institute of Otology, Lempert Research Foundation, Inc.

ally with various means to prevent osteogenetic closure of the fenestra, in a letter dated April 12, 1938, read by T. J. Harris at the American Otological Society, stated as follows: "It is possible to obtain permanently open fistulas of the labyrinth, but up till now this has been the case only occasionally with the material of Sourdille's and mine, and there is not any method known of which is leading to the goal."

Twelve years have gone by since Sourdille and Holmgren made the above quoted statements. Anywhere from 20,000 to 25,000 fenestration operations have since been performed by otologists. Clinical evidence of osteogenesis taking place in the newly created window is no longer apparent four, six or ten weeks later as Sourdille reported. Instead, evidence of impending bony closure of the fenestra very rarely manifests itself before six months postoperatively. The hearing, in those cases wherein osteogenesis does take place, as a rule does not begin to show signs of recession before twelve months postoperatively and sometimes not before eighteen months of good hearing has been enjoyed by the patient. Permanently open labyrinthine fenestras, which up until April, 1938, were only obtainable occasionally with the material of Holmgren and Sourdille, are now in 1950 obtainable in about 75% of all patients operated upon. The obvious progress made in the surgical treatment of otosclerosis since the reports of Holmgren and Sourdille in 1937 and 1938 began following my development and description of the practical one-stage fenestration technic in 1938, which made this surgery practical for both patient and otologist, and continued by leaps and bounds ever since my subsequent development and description of the Lempert fenestra novovalis technic in 1941, whereby the fenestra was moved forward over the surgical dome of the vestibule.

Always remaining mindful of Sourdille's plea of 1937, that we must still work to solve this problem of osteogenesis, I have never relaxed in my efforts to solve this problem and if possible to remove it once and for all as a factor contributing to failure of this surgery. Since 1938 I have been revising all my failures which were clinically suspected as having resulted from osteogenetic closure of the fenestra in order to determine if possible what happened repeatedly and consistently in the fenestra region which could be held responsible for the window having closed either partially or completely, and what can be done to prevent it.

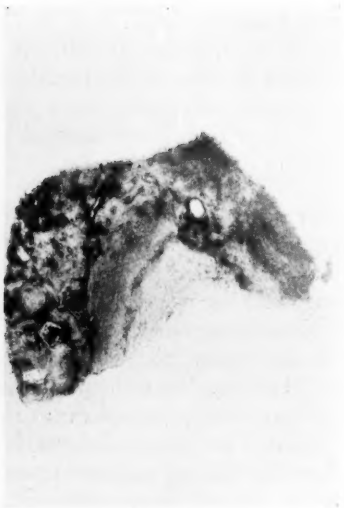

Fig. 1.—Photomicrograph: Otosclerosis of bony capsule of external semicircular canal.

As a result of careful and detailed study of the entire fenestra region during the performance of 517 revisions of osteogenetically closed fenestras in the last twelve years, the following observations were made:

1. At no time following a meticulously performed fenestration operation was osteogenesis observed to have taken place on the injured outer convex surface of the bony capsule of the labyrinth in the region surrounding the fenestra gap after separating and elevating the firmly adherent to it tympanomeatal flap. This observation held true whether the outer surface consisted of periosteal bone only or enchondral bone only.

2. Only following fenestration in fulminating malignant otosclerosis where the bony capsule of the external semicircular canal showed grossly evidence of the disease on the operating table, did I observe during revision of the fenestra, osteogenesis to have simultaneously started and taken place within both the outer surface of the bony capsule and the fenestra rim margin. Since employing the bone-

Fig. 2.—Photomicrograph: Otosclerosis of bony capsule of external semicircular canal.

dust-free cupola technic for creating the fenestra I am able to remove the endosteal bony cupola intact and having it histologically examined. Histological examination of two such bony cupolas has revealed the presence of otosclerosis in the bony capsule of the external semicircular canal (Figs. 1 and 2).

3. Osteogenesis has always been found to have taken place only in the lateral walls of the bony margin of the fenestra rim facing and surrounding the fenestra gap within the bony labyrinthine capsule, whether that fenestra rim margin consisted of periosteal, enchondral and endosteal bone, enchondral and endosteal bone only, or endosteal bone alone.

4. Whenever bone regeneration was observed as having taken place in the lateral walls of the fenestra rim, and as having closed the fenestra gap, it was always found to have started in the endosteal bony layer of the fenestra rim and was as a rule observed to have remained limited to this layer.

This observation held true in revisions of patients operated upon with my first described technic of 1938 when the bony fenestra rim

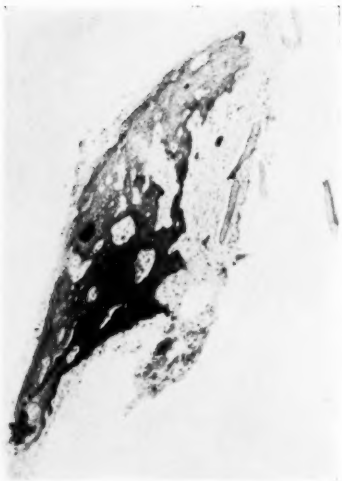

Fig. 3.—Photomicrograph: A newly regenerated endosteal bony lid.

of the trough-like fenestra consisted of all the three histologic layers of the otic capsule, and did not differ in the revisions of patients operated upon with the fenestra nov-ovalis technic of 1941 when I abandoned making the fenestra trough-like, and the fenestra rim consisted of the enchondral and endosteal layers only.

It is quite obvious in view of the above mentioned observation, that removing the periosteal layer and limiting the fenestra rim to the enchondral and endosteal layers does not prevent the onset and progress of osteogenesis in the endosteal layer and its terminating in fenestra closure.

In my article published in the Archives of Otolaryngology, May, 1940,[3] I stated as follows: "From my observations during revisions of the fenestra, I was also forced to conclude that new bone regeneration within the fenestra begins not in the periosteal but in the endosteal layer of the bony capsule."

In support of this statement I published[3] the first photomicrograph of a newly regenerated endosteal bony lid which was removed

during revision of an osteogenetically closed fenestra following fenestration (Fig.3).

Lindsay,[4] after analyzing his histologic observations in the fenestrated Rhesus monkey arrived at the same conclusion and stated in the 1947 issue of the Archives of Otolaryngology as follows: "Failure to maintain an open fistula was in most cases due to the osteogenetic process which took origin from the endosteal surface at the margins."

In the hope of preventing osteogenetic closure of the fenestra I advocated and used gold burnishing burrs for highly polishing the lateral walls of the fenestra rim. In the May, 1940, issue of the Archives of Otolaryngology,[3] I stated as follows:

"The purpose of employing the 24 carat gold burnishing burr is to reduce still further the possibility of bone regeneration in the occasional case by highly polishing the lateral bony walls of the fenestra and simultaneously implanting fine particles of gold within these walls."

After obtaining histologic evidence following controlled experimentation in the Rhesus monkey,[5] that only burnishing the bony margins of the fenestra rim with pure lead to the exclusion of burnishing with all other metal polishing burrs prevented osteogenetic closure of the fenestra in two Rhesus monkeys, I abandoned the use of the gold burr and advocated and used lead burnishing of the fenestra rim in the hope of preventing osteogenetic closure of the fenestra in the human.

5. Following carelessly performed fenestration surgery the study of the osteogenetically closed fenestra region during revisions revealed that bone particles left behind in the fenestra region, or left attached to the portion of the tympanomeatal flap which is destined to cover the fenestra region, can stimulate osteogenesis in any one of the injured histologic layers of the bony capsule with which it comes in contact.

The fact that neither one of the three histologic layers of the fenestra region is immune to osteogenesis under such favorable conditions, has been amply demonstrated experimentally in the Rhesus monkey by Fowler, Lindsay, Shambaugh, Meltzer and myself.

In making the fenestra with the electrically driven polishing burr, bone-dust, which is a by-product of this technic when not com-

Fig. 4.—Photomicrograph: Newly formed bony mass stimulated by bone dust left behind in fenestra region.

pletely removed from the region of the fenestra, will stimulate osteogenesis. Having recognized this fact, I have advocated its removal. In a paper read November, 1939, at a meeting of the New England Oto-Laryngological Society in Boston, which was published in the Archives of Otolaryngology,[3] I stated as follows: "The meticulous removal of all particles of bone-dust from the newly created fenestra is an essential feature in the technical scheme for preventing osteogenesis."

Shambaugh, Jr.,[6] in a discussion of Meltzer's paper in Minneapolis in February, 1940, stated: "For some time I was at a loss to explain the large proportion of closures. Numerous visits were made to Dr. Lempert and as the result of observations made by him and experimental work by Dr. E. P. Fowler, Jr. and others, the importance of bone dust in the closure of the fistula has become increasingly apparent."

During my address,[7] March 20, 1940, before the combined meeting of the Sections on Otolaryngology of the New York Academy

of Medicine, and the Philadelphia College of Physicians, I showed the first photomicrograph (published in the April, 1941 issue of the Laryngoscope) of a newly formed bony mass removed from the fenestra gap during the revision of an osteogenetically closed fenestra which was stimulated by bone-dust left in the fenestra gap during the process of creating the fenestra with the polishing burr (Fig. 4).

In 1947, Holmgren[8] stated as follows: "There is no doubt that the use of the finishing burr and the irrigation and suction of the bone-dust formed plays a considerable role in keeping the fenestra open. Lempert operated with Gullstrand-Zeiss magnifying glasses. Simson Hall and Shambaugh and many others with a microscope. I consider that Gullstrand's loupe is perfectly satisfactory, and it appears to me to be of minor importance which magnification is used. Simson Hall and Shambaugh operate under continuous irrigation and suction; Lempert with repeated irrigation and suction. There is little difference."

However, every otologist acquainted with fenestration surgery knows that whereas the removal of bone-dust is essential, its complete absence will not prevent fenestra closure resulting from the naturally existing tendency for osteogenesis within the freshly injured bony lateral walls of the fenestra rim. When the fenestra was made with the hammer and chisel without the creation of bone-dust almost all fenestras eventually closed as a result of osteogenesis.

6. Osteogenesis has been observed to have taken place within the perilymphatic space of the external semicircular canal.

Histologic investigation of post-fenestrated Rhesus monkeys[5] has demonstrated that such endosteal perilymphatic osteogenesis was usually the result of bone-dust or bone chips having been unavoidably lost within the perilymphatic space during the pulverization of the endosteal bony layer when creating the fenestra with the polishing burr.

Lindsay[4] stated in the November, 1947 issue of the Archives of Otolaryngology as follows: "The histologic examination has demonstrated that although at operation the technic appeared to be carried out faultlessly, the complete removal of bone-dust and fragments was rarely accomplished."

Because of this observation I have, in 1947, developed and since employed the cupola technic[9] for creating the bone-dust-free fen-

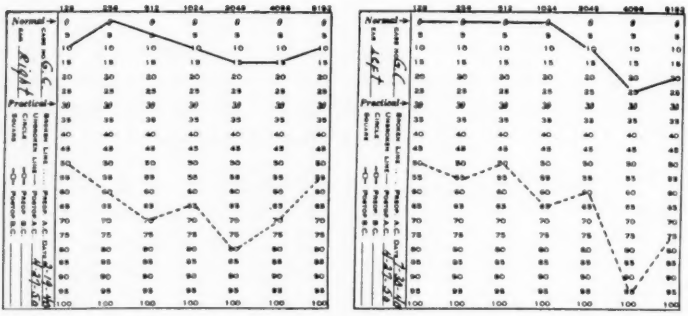

Fig. 5.—Audiograms: Preoperative and postoperative audiograms of right and left ears of G. C.

estra. Since no bone-dust is created with this technic, bone particles can no longer be unavoidably lost within the perilymphatic space and stimulate perilymphatic endosteal osteogenesis.

Since my development and employment of the bone-dust-free cupola technic for the creation of the fenestra nov-ovalis, the problem of removing bone-dust from the fenestra gap has been disposed of. Though perilymphatic osteogenesis has not been encountered since the adoption of this technic, osteogenetic endosteal closures of the fenestra rim did however occur in some cases and were observed during revisions. This proves again that though osteogenesis is hastened by the presence of bone-dust it is not prevented by absence thereof.

7. Fenestra closure was observed to have taken place much faster and much more completely in the region posterior to the ampulla of the external semicircular canal. In patients who gave a positive response to the fistula test, despite the fact that the postoperative hearing improvement had already receded to a level no longer practical, the fenestra was always observed to be closed except in the region closest to the ampulla of the external semicircular canal.

In November, 1939, in a paper read before the New England Oto-Laryngological Society in Boston, and published in the Archives of Otolaryngology, May, 1940, issue, under heading of "Placement of the Tympanomeatal Membrane," I took cognizance of the neces-

sity for preventing blood from entering the perilymphatic space and stated as follows:[3] "Adjustment and placing of the tympanomeatal membrane and sealing of the newly reconstructed tympanic cavity (including the fenestra in the external semicircular canal) must not be undertaken until all bleeding has ceased and all accumulated blood has been removed from within the reconstructed cavity."

However, I have never failed to recognize the fact that despite such meticulous technic some blood still entered the 0.8 mm perilymphatic space of the external semicircular canal and if not quickly absorbed it can form a connective tissue matrix underneath the flap which could encourage osteogenesis within the lateral bony walls of the fenestra rim.

The above stated recognized fact plus the observation made during revisions, led me to suspect that osteogenetic closure is slow and incomplete in the region of the ampulla of the external semicircular canal as compared to the region posterior to the ampulla, because the perilymphatic space in the region of the ampulla, being much deeper and wider, favors the dissemination and absorption of blood much more than the shallow and narrow perilymphatic space in the region posterior to the ampulla.

By following this line of thought, it occurred to me that perhaps by removing the incus and moving the fenestra forward over the surgical dome of the vestibule where it will communicate directly with the perilymphatic space of the vestibule, which is 3 mm wide and 5 mm deep, the dissemination and absorption of blood in the perilymphatic space would be greatly facilitated and would reduce the incidence of formation of a fibrous tissue matrix which promotes bone regeneration in the fenestra gap. Thus, the fenestra nov-ovalis technic was born.

The Lempert fenestra nov-ovalis technic, which I have described in the 1941 issue of the Archives of Otolaryngology, has since been adopted universally and credited by otologists everywhere as being the one great single factor which has to date contributed most towards the prevention of osteogenetic closure of the newly created fenestra.

In 1946, Altmann[10] stated as follows: "According to Lempert, the tissue proliferation takes place mainly in response to the blood entering the perilymphatic space in the region of the fistula. In

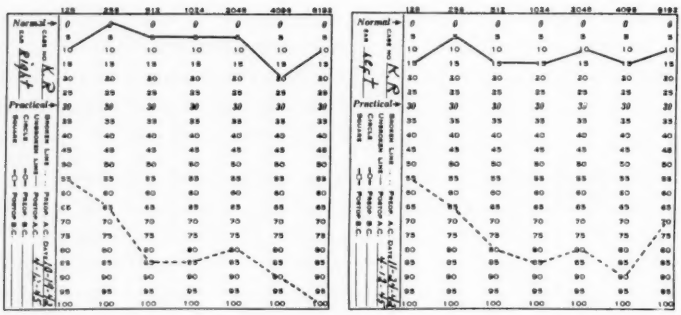

Fig. 6.—Audiograms showing preoperative and postoperative hearing in right and left ears of K. R.

a large space the masses of blood are more easily disseminated, absorbed and eliminated than in a small space. In the latter they are more likely to remain and stimulate tissue proliferation. In support of this opinion, Lempert mentioned his observation that in operative fistulas which extend forward well over the ampulla excessive tissue proliferation leads to bony closure only in the posterior part. The part which overlies the narrow canal, not in the anterior part which overlies the wider ampulla. The findings in case three of Preuss, in which the fistula of the lateral semicircular canal was closed with bone in the posterior half, but only with connective tissue in the region of the ampulla, are in good agreement with Lempert's observations. In case of Ruttin a fistula had remained partially open thirteen years after tympanomastoidectomy for cholesteatoma. Here again the posterior part was closed with hyperostotic bone while the anterior part close to the ampulla contained only connective tissue."

On the other hand, Sourdille,[11] in 1949, criticized the Lempert fenestra nov-ovalis technic and stated as follows:

"My very distinguished colleague, Dr. Lempert, has taken the heavy decision to remove the incus. The immediate consequence of such removal of the incus was that the operative indications for his procedure found themselves considerably reduced. His procedure could no longer be applicable except in the slightly or moderately deafened."

"The conservation of the incus, on the contrary, strengthens the conduction of the high frequencies and enables a gain of 30 to 40 decibels for cases of progressive deafness bordering upon total deafness."

As an illustration that the removal of the incus as practiced with the fenestra nov-ovalis technic is not damaging to the end result desired, and can enable a gain of much more than 40 decibels in both the low and the high frequencies for cases of progressive deafness bordering upon total deafness, I submit for your consideration some of the end results obtained in extremely deafened patients despite the removal of the incus.

I wonder if my colleague, Dr. Sourdille, believes that if the incus were retained a better hearing improvement could have been obtained in these patients. One of these patients had the right ear operated upon with the incus left in position and the left ear with the incus removed.

As a result of the observations made during revisions of osteogenetically closed fenestras and some of the measures instituted for the prevention of such closures, which were based upon these observations, closure of the fenestra due to osteogenesis has been reduced to approximately 25% of all patients operated upon. It is nevertheless a non-disputable fact that many fenestras which some otologists clinically believe to be patent are in fact osteogenetically closed. If any otologist doubts this statement let him revise and inspect the fenestra in those cases which he considers and reports as successful though not practical, wherein there has been an 11-15 decibel improvement maintained for over a period of two years after receding from an original postoperative improvement of 30 decibels or more, and he will be shocked to find that bony closure of the fenestra exists. Whether the window is completely or partially closed will depend upon how soon after two years postoperatively he revises that fenestra. If he revises an ear in which a 50% drop in the postoperative decibel improvement has been observed, he will invariably find osteogenetic closure even though the hearing is still 15 or 20 decibels higher than preoperatively in each of the speech frequencies. It is also a well established fact that no matter how skillfully and meticulously the fenestra nov-ovalis technic is equally carried out in a series of cases, some fenestras eventually close and others remain permanently patent. Furthermore, none of us have ever been able to

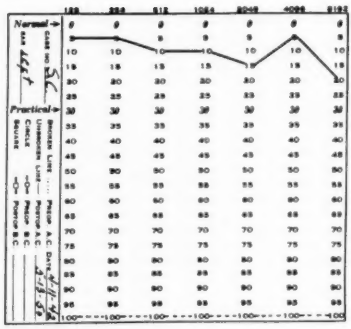

Fig. 7.—Audiogram showing preoperative and postoperative hearing in left ear of S. C.

predict which fenestra will remain permanently patent. Neither were we able to identify with any degree of certainly which of the various technical factors employed for the prevention of osteogenesis was really responsible for a fenestra remaining permanently patent.

Was it the burnishing with gold that made it possible for more fenestras to stay open with the fenestra nov-ovalis technic? Evidently not, since I used it with both technics and also since windows were found open with or without the use of gold burnishing.

Was it the removal of bone-dust which was solely responsible for this fact? Evidently not, since I meticulously removed bone-dust with the use of both technics. Furthermore, fenestra closures were observed also following the use of the bone-dust-free cupola technic.

Was it because the chances for dissemination and absorption of blood entering the perilymphatic space were enhanced with the nov-ovalis technic, and made it more difficult for a connective tissue matrix to form and encourage osteogenetic repair of the fenestra rim, that more fenestras remained patent?

Was it enchondralization that prevented fenestra closure? Evidence suggests otherwise. Fenestras closed with or without enchondralization as a result of endosteal bone regeneration of fenestra rim. Osteogenesis as a rule does not occur on outer surface of bony cap-

sule whether capsule is enchondralized or not when the fenestra is sealed with the tympanomeatal flap.

Was it gold-burnishing that always prevented osteogenesis on the outer surface of the fenestra? If so, why did it not prevent it in the lateral walls of the fenestra rim?

Was it the fact that the fenestra made with the fenestra nov-ovalis technic was wider than the fenestra made with the formerly employed technic, and, therefore, reduced the incidence of fenestra closure? Then why did some of the widest made fenestras close?

Why did the outer surface of the fenestra region regardless of which bony histologic layer was removed or not removed, not show signs of regeneration as a rule, no matter which technic was used and regardless of whether that bony surface was burnished or not burnished with gold.

However, a careful analysis of all my observations made during my research study of the osteogenetically closed fenestras following meticulously executed labyrinthine fenestration in the Rhesus monkey, and the revision of 517 osteogenetically closed fenestras following the meticulously performed fenestration in the human gave me the impression that the key to the mystery of deliberately preventing osteogenesis within a newly created labyrinthine fenestra lies somewhere in the investigation of the possibility of some existing relationship between the following three observations made:

1. In the Rhesus monkey when the fenestra was not sealed with the tympanomeatal flap, when osteogenesis took place it did so both outside the fenestra rim and within the fenestra rim margins.

2. When the fenestra was sealed with the tympanomeatal flap, osteogenesis took place as a rule only in the endosteal margins of the fenestra rim, and not on the outer bony surface surrounding the fenestra gap. This observation held true in both the human and the Rhesus monkey. It also held true whether the fenestra region was burnished or not burnished with gold or lead.

3. Since the universal adoption of the fenestra nov-ovalis technic in the human, whereby the fenestra was moved forward over the surgical dome of the vestibule, the incidence of osteogenesis in the endosteal layer of the fenestra rim margins has been greatly diminished.

The more I thought about these three observations the more convinced I became that if the secret of the prevention of osteogenesis within the fenestra rim is ever to be solved a carefully conducted study of the human fenestras which remained permanently patent because osteogenesis did not take place will have to be made. By comparing the findings in the osteogenetically closed fenestras with the findings in the permanently free from osteogenesis fenestras I hoped to find the clue to the prevention of osteogenesis within the fenestra rim margins which could point the way to the development of a technic for keeping deliberately every fenestra permanently patent.

I decided to investigate a series of permanently open fenestras and observe if possible what does happen when osteogenesis does not occur in fenestra rim margins and the fenestra stays permanently open, and what does not happen when osteogenesis does occur and either complete or partial fenestra closure is observed at time of revision. What if anything did uniformly and consistently happen when the fenestra remained permanently and widely patent?

Since 1947, I have revised and carefully studied the findings in the fenestra region of 100 patients whose hearing following the fenestra nov-ovalis operation either (a) never improved; (b) receded still further; or (c) audiometrically improved too little to be noticeable by the patient.

In 517 of the 617 revisions which I performed to date I was mainly interested in studying the observations made in the osteogenetically closed fenestras. In this special series of 100 at no time improved ears following the fenestra nov-ovalis operation my special attention was diverted to the careful study of those fenestras in this series which showed no signs of osteogenesis having taken place and, therefore, remained permanently patent.

Of these 100 ears the fenestra was found to have undergone endosteal osteogenetic repair of the fenestra rim in 28, which resulted in complete closure of 22 fenestras and in incomplete closure of the fenestra in six ears at the time of revision. The outer surface of the otic capsule surrounding the fenestra gap and to which the flap was adherent showed no signs of osteogenetic repair in this region in any of these 28 otherwise osteogenetically closed fenestras. In 72 ears osteogenesis did not take place anywhere in the fenestra rim, and the fenestra gap was found widely and completely patent.

The following are the observations which were made within the 72 ears wherein the fenestra remained permanently patent because osteogenesis did not take place:

After separating the tympanomeatal flap from the outer bony surface of the labyrinthine capsule surrounding the fenestra gap to which it was firmly adherent, complete absence of bone regeneration was noted in this extramarginal fenestra rim region. The cutaneous flap was readily separated from the bony capsule to which it was firmly adherent until the very margin of the fenestra rim was reached. At the margin of the fenestra rim the inner surface of the flap resisted easy separation from its attachment, and required careful separation from its adhesions because it was invaginated trough-like into the fenestra gap slightly beyond the fenestra rim and was in contact with the perilymphatic space. Its side walls were firmly adherent to the lateral bony walls of the fenestra rim margin. After having gently separated the flap from its adhesion to the bony fenestra rim and elevating it, inspection of its inner surface which was adherent to the bone revealed a definitely moulded impression of the fenestra gap in the precise region of the portion of the flap which became invaginated into the perilymphatic space and beyond the fenestra rim. The endolymphatic labyrinth was observed to have remained unchanged and apparently normal with no visible signs of having been adherent to the tympanomeatal flap discernible in 68 of the 72 ears. Not the slightest sign of osteogenetic repair was observed to have taken place either extramarginally on the outer surface of the otic capsule surrounding the fenestra rim, or intramarginally in the fenestra rim proper in any of the 72 ears.

In four of these 72 ears, the perilymphatic space was partially filled with fibrous tissue and the labyrinth was no longer translucent but milky white in appearance and sclerotic in texture. I gained the impression from the observations made in the perilymphatic space that the flap must have been adherent to the fibrous tissue surrounding the labyrinth, but not to the labyrinth proper in these four fenestras since I succeeded in lifting the flap out of the perilymphatic space without pulling the labyrinth out with it.

In all of these 72 fenestras which have been found to have remained completely patent for periods varying from one to eight years postoperatively, osteogenesis has been completely prevented.

The postoperative time of the 72 open windows encountered in this series of 100 revisions:

1 year	11 ears	4 years	9 ears
18 months	9 ears	5 years	6 ears
2 years	22 ears	7 years	2 ears
3 years	12 ears	8 years	1 ear

When one compares the most important observations made in the osteogenetically closed fenestras with those made in the permanently patent fenestras, it becomes quite obvious that the role played by the tympanomeatal flap which is used for sealing the fenestra is the determining factor between the occurrence and nonoccurrence of osteogenetic closure of the fenestra. The most outstanding differentiating observations made between the osteogenetically closed fenestra and the permanently patent free from osteogenesis fenestra was: in the osteogenetically closed fenestra the inner surface of the tympanomeatal flap was found to be smoothly adherent only to the outer bony layer of the otic capsule surrounding the fenestra gap extramarginally, while the fenestra gap was intramarginally obliterated by new bone growing from the bony margins of the fenestra rim; whereas in the permanently patent fenestra, the tympanomeatal flap was found to be adherent both to the otic capsule extramarginally to the fenestra gap and intramarginally to the fenestra rim with the fenestra gap obliterated by the invaginated portion of the flap instead of by new bone. Thus, it can be readily seen that whenever physical impedance to osteogenesis was created by the accidental invagination of the flap into the fenestra gap and its adhesion intramarginally to the fenestra rim the fenestra stayed permanently open. Also that whenever invagination of the flap into the fenestra gap did not take place physical impedance to osteogenesis within the fenestra rim did not occur and the fenestra closed either wholly or partially by osteogenesis (Figs. 8*a* and 8*b*).

An analysis of these above mentioned facts can lead only to the logical and scientific conclusion that unless invagination and intramarginal adhesion of the tympanomeatal flap to the bony fenestra rim takes place osteogenetic closure of the newly created fenestra cannot as a rule be prevented. Adhesion to the outside of fistula

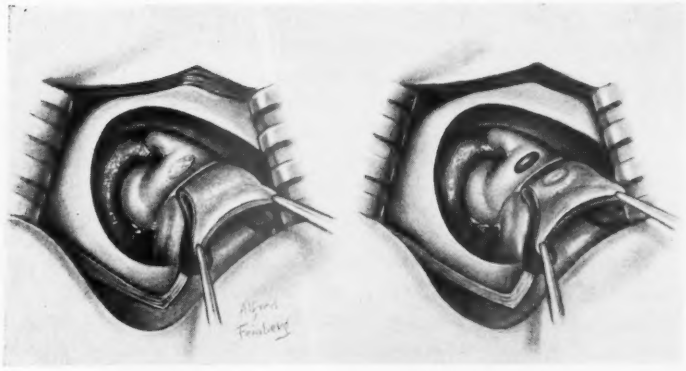

Fig. 8*a*.—Diagrammatic drawing showing the smooth inner surface of the tympano-meatal flap, as seen during revision in all the osteogenetically closed fenestras.

Fig. 8*b*.—Drawing showing the moulded impression of the lateral bony walls of the fenestra rim margins on the inner surface of the tympano-meatal flap secondary to its invagination into the fenestra gap, as seen during revision in all the permanently patent fenestras wherein osteogenesis did not take place.

only, cannot prevent osteogenesis from taking place within the fenestra rim.

If a method of always assuring invagination and adhesion of the flap to the bony fenestra rim margins could be devised, osteogenetic closure of the fenestra would be done away with.

That soft-tissue impedance could prevent osteogenetic closure of the fenestra, though only proven in 1950, was nevertheless anticipated in 1938, can be seen from the following statement which I had then made. In describing the first one-stage fenestration technic for the improvement of hearing in otosclerosis in the July, 1938, issue of the Archives of Otolaryngology under the heading "Insertion of Tympanomeatal Cutaneous Membrane into the Troughlike Fenestra of the External Semicircular Canal"[12] I stated: "A piece of paraffin mesh rolled into a small ball is placed over the portion of the tympanomeatal membrane which covers the outer aperture of the troughlike fenestra in the external semicircular canal. After

gentle pressing and molding of the paraffin mesh over Shapnell's membrane, the latter is inserted into the troughlike fenestra so as to line the inner surface of its walls, extend immediately beyond the edge of the inner aperture of the fenestra, and lie in direct contact with the perilymph. The paraffin mesh ball is held in place by additional pieces of paraffin mesh inserted into the mastoid wound. On the care and precision with which this step in the technic is carried out depends the permanent maintenance of the fenestra in the bony capsule."

Being cognizant of the fact that the 0.8 mm width of the perilymphatic space of the external semicircular canal in the region posterior to the ampulla was neither adequate enough to permit the creation of a fenestra nor wide enough to permit the invagination of the tympanomeatal flap into the fenestra gap beyond the endosteal bony layer of the fenestra rim and into the perilymphatic space; also when taking into consideration that the elevated tent-like position of the incus in its relation to the position of the external semicircular canal fenestra on a much lower level will interfere with the insertion of the flap into the perilymphatic space and beyond the fenestra rim, I decided to make a trough-shaped fenestra so that the circumference of the outer margin of the fenestra rim will be wider than the naturally limited width of its perilymphatic margin in order to facilitate the invagination of the flap into the fenestra gap.

However, after developing in 1940 and employing the fenestra nov-ovalis technic exclusively since 1941, I no longer made the fenestra trough-shaped because I felt that by removing the incus and making a wider fenestra, intramarginal insertion of the flap into the fenestra gap would be greatly facilitated. In my discussion in 1947,[13] of the measures I have instituted for the prevention of osteogenesis, I again stated under the heading of "Soft Tissue Impedance" as follows: "When covering and sealing the newly created fenestra I pressed the tympanomeatal membrane tightly into the fenestral gap in the hope of physically preventing osteogenesis of the bony rim from spreading and subsequently closing the gap."

Both my 1938 and 1947 statements mentioned above were expressions of my belief in the possibility of preventing osteogenetic closure of the fenestra by invaginating the portion of the tympanomeatal flap facing the fenestra into the fenestra gap beyond the innermost bony layer of its rim and into the perilymphatic space so that

it would adhere to the bony margins of the fenestra rim. This belief in turn was based upon the observations which I have made during the healing process within the postoperative mastoid wound for a period of 20 years of active practice in the art of otologic surgery prior to 1938.

These observations were as follows: 1. In my practice of otologic surgery I was often called upon to operate for recurrence of infection within previously mastoidectomized ears. I often found almost the entire outer mastoid cortex intact and the interior of the mastoid process full of diseased mastoid cells. Being young in experience and observation, I considered such findings following a previously performed mastoidectomy by an otologist other than myself as evidence of incompletely performed surgery on his part. However, as time passed I was called upon to perform secondary mastoidectomies on some of my own previously mastoidectomized patients in whom I had definitely removed the entire outer mastoid cortex and every single cell within the confines of the mastoid process. When I found to my chagrin that regeneration of both cortex and cells had taken place in my own previously mastoidectomized ears as well, I was shocked into a more benevolent and sympathetic evaluation for the surgical efforts of those other otologists whose mastoidectomized patients I was reoperating.

2. Early in my career of otologic surgery and before the advent of antibiotic therapy, the postoperative, postauricular mastoid wound following the simple mastoidectomy was left wide open to continuously maintain drainage of the infection, and was packed with iodoform gauze to promote healing by granulation. Whenever, during the course of such wound treatment, the postauricular incised skin edges happened to become invaginated into the mastoid wound a permanent postauricular fistula resulted.

When surgically correcting permanent postauricular fistulas following surgery for acute suppurative mastoiditis, I observed that the extent to which the outer mastoid cortex regenerated always coincided with the point at which the incised postauricular skin and periosteum happened to become invaginated into the postopertive mastoid cavity.

Based upon the above mentioned observations I felt that if following the meticulous performance of the fenestration operation osteogenesis is to be prevented and the fenestra is to remain per-

manently patent as originally created, then intramarginal invagination of the inner surface of the flap into the fenestra gap is essential and stated so in my original paper published in 1938.

However, scientific proof of the correctness of this theory of mine was only obtained after observing during revisions that in every one of the osteogenetically closed fenestras the tympanomeatal flap was found not to be invaginated into the fenestra gap and osteogenesis has taken place intramarginally in the fenestra rim, and that in every one of the permanently patent fenestras the tympanomeatal flap was always seen invaginated into the fenestra gap and adherent intramarginally to the fenestra rim and osteogenesis has not taken place.

That the secret of obtaining a patent fenestra following the fenestration operation may be some form of soft-tissue-impedance to the start and progress of osteogenesis has been suspected by other otologists, becomes quite evident from some of the theories on this subject advanced in the otologic literature since 1938.

Campbell[14] believed that osteogenetic closure of the fenestra could be prevented by adhesion of the flap to the endosteum. In reporting his postoperative results in 30 cases following the use of my first described one-stage fenestration technic, Campbell stated in the November, 1941, issue of the Laryngoscope as follows: "It seems to me more logical to believe that there is less danger of the fistula closing either by bony or fibrous tissue if the periosteal-endothelial lining of the semicircular canal is not removed or broken during the fenestration process, because then the raw surface of the skin flap deflected from the auditory canal wall to cover the fistula comes into close contact with the raw surface of the endosteum and makes a primary union before there has been time for bone regeneration to cover this fenestrated area."

In an attempt to reduce the percentage of failures caused by postoperative labyrinthitis, I tried and succeeded in leaving the endosteum intact in 100 cases. In the January, 1945, issue of the Archives of Otolaryngology,[15] I reported as follows:

"As a result of such treatment of the newly created fenestra, the severity of the postoperative labyrinthitis and the freqency of damage of the cochlear nerve were considerably reduced. The improvement of hearing in these cases reached as high a level as in the

other cases in which the endosteum was removed. However, it was unfortunately observed in this series of cases that both the frequency and the rapidity of osteogenetic closure of the newly created fenestra were definitely enhanced when the endosteum was left intact."

Lindsay[16] after observing histologically adhesion of the labyrinth to the tympanomeatal flap in some of the fenestras which he found patent following experimental fenestration of the labyrinth in the Rhesus monkey, was of the opinion that this form of soft-tissue impedance could prevent osteogenetic closure of the fenestra, and stated in 1946 as follows: "The evidence suggests that any fistula remains open only by virtue of the union which occurs between the membranous labyrinth and the covering flap to the exclusion of osteogenetic tissue."

I[7] was the first to call attention to the fact that adhesion between the tympanomeatal flap and the endolymphatic labyrinth has taken place in the presence of a 14 day old open labyrinthine fistula in the human, and published the photomicrograph of a sectioned postfenestrated human temporal bone in 1941 showing same. I, nevertheless, was forced to disagree with the conclusion which Lindsay had drawn in 1946 as a result of his having made the same observation which I had made in both human and monkey that bone regeneration can take place underneath the flap in the endosteal layer of the fenestra rim in the presence of adhesion of the membranous labyrinth to flap and gradually separate the two, Lindsay's conclusion appeared to be illogical, and I[13] stated in 1947 as follows: "The evidence suggests that any union which occurs between the membranous labyrinth and the covering flap to the exclusion of osteogenetic tissue occurs only by virtue of the fistula's remaining open."

In support of my statement I[13] published a photomicrograph of the sectioned temporal bone of a postfenestrated Rhesus monkey which showed bone regeneration taking place in the endosteal bony layer of the fenestra rim and growing knife-like across the fenestra gap and separating the membranous labyrinth from the tympanomeatal flap to which it had become adherent (Fig. 9 and 10).

Lindsay[4] after further study of the labyrinthine fistulas in the Rhesus monkey wherein the membranous labyrinth appeared to be adherent to the tympanomeatal flap stated in 1947 as follows: "In no case had bone been completely prevented from being deposited beneath the flap."

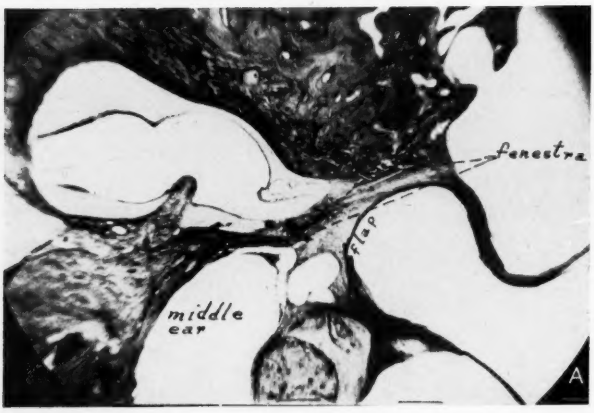

Fig. 9.—Low Power Photomicrograph: Endosteal bone growing and, knife-like, separating the labyrinth from the flap to which it became adherent. Exp. III. No. 7.

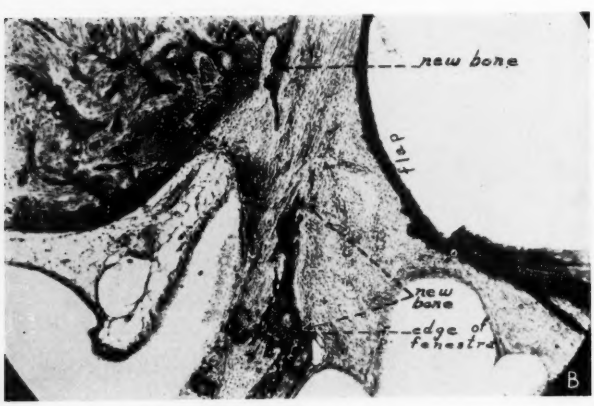

Fig. 10.—High Power Photomicrograph.

This observation of his is sufficient proof that adhesion of labyrinth to the flap in the human even if it does occur does not prevent osteogenesis from taking place in the fenestra rim and, therefore, cannot be depended upon to prevent ultimate osteogenetic closure of the fenestra. Once osteogenesis is permitted to start there is no way of knowing when and where it will stop. Invagination of the tympanomeatal flap into the fenestra gap is to date the only definitely proven scientific means of preventing osteogenesis from starting and thus assuring a permanently open fenestra.

Experience with revisions has taught me that if some of Lindsay's monkeys showing partial osteogenesis of the fenestra rim underneath the flap would have been permitted to live a little longer, he would have in most instances observed that separation of the flap from its adhesion to the labyrinth had taken place as a result of the further progress made by the osteogenetic process in its mission of fenestra closure. The following photomicrograph of the sectioned temporal bone in the postfenestrated Rhesus monkey shows bony closure having separated the flap from the labyrinth and giving the impression of adherence of the labyrinth to the newly grown bone (Fig. 11).

I believe that if Lindsay will re-examine his histologic material he will observe that whenever osteogenesis did not take place and the window remained widely patent in the presence of adhesion of the flap to the membranous labyrinth, invagination of the inner surface of the flap and its adhesion to the endosteal bony layer of the fenestra rim margin has also taken place. He will also observe that wherever intramarginal adhesion of the flap to the fenestra rim did not take place, even though both extramarginal adhesion of the flap to bone and adhesion of flap to the labyrinth is present, osteogenesis of the fenestra rim has taken place and narrowed the fenestra.

Shambaugh Jr.[17] believes that soft tissue impedance to osteogenesis within the fenestra gap could be created by endosteum-hemming the fenestra rim and stated in 1949, as follows:

"As far as possible the fragments of the endosteal membrane are folded over the sharp knife-like edges of the completed fenestra, so that the fenestra is endosteum-hemmed."

The fact is that not much endosteum-hemming is ever possible. Holmgren attempted to cover the fenestra edges with endosteum many years ago and found it wanting.

Fig. 11.—Photomicrograph of fenestrated Rhesus monkey.

The evidence suggests that invagination of the viable tympanomeatal flap into the fenestra gap and its intramarginal adhesion to the bony fenestra rim is the secret in the prevention of osteogenesis arising from the rim margins and consequently, in the prevention of osteogenetic closure of the fenestra gap.

When invagination of the flap occurs nothing else is necessary. When invagination does not take place nothing else matters as a rule.

Why has the percentage of permanently patent fenestras risen so dramatically since the adoption of the fenestra nov-ovalis technic? Holmgren[8] in 1947, stated as follows: "It is evident that Lempert's method has conquered, and there is a certain interest in explaining why this is the case."

"The most important point is the following: the new fenestration is made on the dome of the vestibule, which involves removal of the incus and, usually, of the head of the malleus. At this site

the new fenestration can be made larger than at any other point in the vestibular apparatus, and the membranous labyrinth is further from the endosteum."

Prior to my carefully conducted study of the findings in the 72 permanently patent fenestras following the fenestra nov-ovalis operation I too believed that the reason osteogenetic closure of the fenestra became less frequent following the use of the fenestra nov-ovalis technic was because in the presence of a wider fenestra and deeper lumen the chances for bone regeneration were lessened, and that if it did take place it would exhaust itself and still leave a fenestra wide enough to permit the retention of the hearing improvement.

Since my observations made within the permanently patent 72 fenestras wherein osteogenesis was prevented from starting, I now am convinced that the reason more fenestras remained permanently free from osteogenetic repair following the fenestra nov-ovalis technic was because accidental invagination of the inner surface of the tympanomeatal flap into the fenestra gap and its intramarginal adhesion to the fenestra rim was favored by the following surgical factors involved:

1. The removal of the incus from its tent-like position, and by moving the fenestra forward over the surgical dome of the vestibule, Both of these surgical factors facilitate invagination of the tympanomeatal flap by favoring the thinnest portion thereof reaching and sealing the fenestra gap.

2. The greater width of the fenestra gap of the fenestra nov-ovalis. This permits invagination of the flap more readily than the much narrower fenestra formerly obtainable in the region posterior to the ampulla of the external semicircular canal.

3. The much greater depth of the perilymphatic space in the region of the fenestra nov-ovalis as compared to the 0.8 mm depth of the perilymphatic space in the region posterior to the ampulla of the external semicircular canal. This permits invagination of the flap without compression of the labyrinth, thus avoiding sequellae which may arise.

4. The thinner fenestra rim margins of the fenestra nov-ovalis, as compared with the thickness of the margins of the trough-shaped fenestra made formerly in the region posterior to the ampulla of the

external semicircular canal. By shortening the distance from the outer bony surface of the fenestra rim margin to the perilymphatic space immediately beyond its innermost endosteal bony surface, less pressure was required for invagination of the flap into the fenestra gap.

Shambaugh[18] believes that thinning of the fenestra margins by removing the periosteal bony layer is necessary for the prevention of osteogenesis outside the fistula, and stated in 1946, as follows:

"Enchondralization of the fistula has virtually eliminated bony closure due to osteogenesis from outside the fistula, as we have observed in fistulas made by this technic that subsequently closed and were reopened."

The observations which I have made during the performance of 617 revisions suggest that enchondralization for the prevention of osteogenesis outside the fistula is not necessary, since adhesion of the tympanomeatal flap to the outer bony surface of the fistula prevents that, whether the fistula is enchondralized or not.

Therefore, I believe that it is more logical to assume that since thinning of the fenestra margins facilitates invagination of the flap into the fenestra gap, enchondralization can in reality become instead a greater asset towards the prevention of osteogenesis inside the fistula.

How can invagination of the viable tympanomeatal pedicle flap into the fenestra gap and its adhesion to the lateral walls of the bony fenestra rim be deliberately so induced that osteogenesis both outside and inside the fistula will always be prevented?

I have always believed in the possibility of preventing osteogenetic repair of the newly created fenestra by effecting adhesion of the tympanomeatal flap to the lateral bony walls of the fenestra rim and stated so, first in 1938 and again in 1947. However, the method I employed for bringing this about was haphazard and inadequate because it was based upon pressure exerted blindly upon the tympanomeatal flap by the packing within the postoperative mastoid wound without ever being able to observe whether the pressure created was correctly directed and resulted in intramarginal invagination of the flap in the region of the fenestra gap. Therefore, whenever invagination of the flap into the fenestra gap did occur

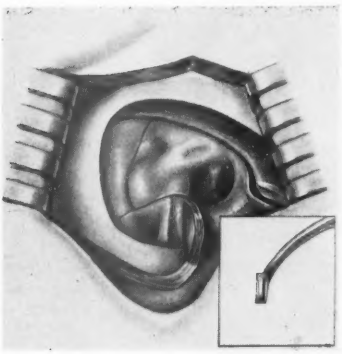

Fig. 12.—Drawing shows tympano-meatal flap invaginated into fenestra nov-ovalis. It also shows the metal invaginator employed for that purpose.

it was accidental and unbeknown to me and by the same token, whenever a fenestra remained permanently patent and not having had any definite knowledge that invagination and adhesion of the flap to the fenestra rim had taken place in such instance, I was in no position to prove scientifically or claim to my own satisfaction that when a certain fenestra has remained permanently open it did so because invagination of the flap did take place.

Today, after revising 72 permanently patent fenestras, I am in possession of definite scientific proof that whenever invagination did occur osteogenesis was prevented. I am, therefore, convinced that by using a technic of directly and visibly invaginating the portion of the tympanomeatal flap covering the fenestra region into the fenestra gap and keeping it invaginated until it becomes and remains intramarginally adherent to the fenestra rim (instead of the blind pressure technic formerly employed) osteogenesis will thus be always prevented from starting, and osteogenetic closure of the fenestra will become a thing of the past.

The technical factors governing the prevention of osteogenesis, both outside and inside the fistula by means of the partly invaginated flap are:

1. Placement of the tympanomeatal flap in position so that its thinnest portion will cover the fenestra gap.

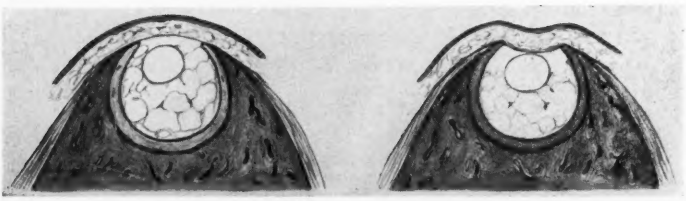

Fig. 13*a*.—Shows cross section of fenestra nov-ovalis covered with the tympano-meatal flap in the traditionally employed manner stretched across the fenestra gap and without covering the lateral walls of the fenestra rim margins.

Fig. 13*b*.—Shows cross section of fenestra nov-ovalis with the tympano-meatal flap invaginated into the fenestra gap and covering the lateral walls of the fenestra rim margins.

2. Invagination of the thin portion of the flap facing the fenestra into the fenestra gap under direct vision and with a specially devised metal invaginator (Figs. 12, 13*a* and 13*b*).

3. Implantation of a cotton inlay, moistened with Ringer's solution, into the trough shaped space created by the invaginated portion of the flap. This induces contact and adhesion of the invaginated part of the flap with the lateral bony walls of the fenestra rim (Fig. 14).

4. Placement of the intramastoid wound dressing so that it will cover the inlay and hold the entire flap and its invaginated portion in the position originally placed. Pledgets of gauze soaked in Ringer's solution are placed on top of the cotton inlay. Dry paraffin mesh gauze is then placed on top of the moist gauze to exert mild pressure within the intramastoid wound.

5. Keeping the intramastoid wound dressing in position long enough to permit adhesion of the flap both extramarginal to the fistula and intramarginally within the fistula. The paraffinated mesh gauze is removed on the seventh postoperative day; the gauze dressing is removed on the ninth day and the cotton inlay on the twelfth day.

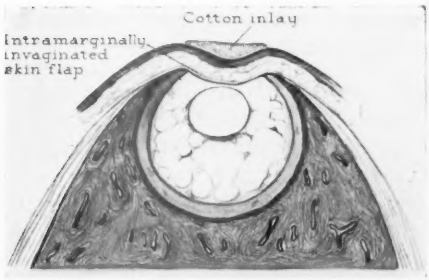

Fig. 14.—Cotton inlay; intramarginally invaginated skin flap.

CONCLUSION

In 1949 Sourdille[11] stated as follows: "For the permanence of the labyrinthine fistula no decisive principle has been advanced since his suggestions made in 1935. Whatever instruments are used for its creation: gouge, burr, scraper, curette; whatever may be the position, the dimensions, the form, the treatment of the osseous section: mechanical, chemical, physical, the ablation of the osteal and endosteal dust, the qualities of the membranous covering—none of these elements is decisive; about 50% of the fistulas shrink or close up after a first opening."

If Sourdille had been acquainted, in 1949, with the results of many otologists who were skillfully performing the Lempert fenestra nov-ovalis operation, I am certain that he would not have made his above quoted pessimistic statement.

In view of my latest contribution towards the understanding of how a fistula remains permanently open following labyrinthine fenestration, and the possibilities offered from now on for keeping all the newly created fenestras open permanently, I hope that my colleague, Dr. Sourdille, will now agree that a decisive principle for the permanence of the labyrinthine fistula has finally been advanced.

Holmgren,[19] in 1937 raised the following questions: "How shall the fistulae be made in order to produce the greatest primary improvement in hearing and how shall they later be handled in order to achieve the greatest permanent good? Those are the two important questions

demanding attention. Once these are answered, one may expect that the surgical management of otosclerosis will be successful in the hands of those who can maintain the necessary asepsis."

Unquestionable evidence is obtainable in the human that; (a) intramarginal adhesion of the tympanomeatal flap to the lateral bony walls of the fenestra rim secondary to its invagination into the fenestra gap prevents the onset of osteogenesis and subsequent closure of the fenestra nov-ovalis; (b) the Lempert fenestra nov-ovalis technic favors invagination and the bony adhesion of the viable tympanomeatal flap.

My theory that invagination of the tympanomeatal flap into the fenestra gap can prevent osteogenetic closure of a newly created fenestra was logically conceived in 1938, scientifically proven since, and found to be practical in its application. I believe that Holmgren's questions of 1937, have finally been answered.

119 East 74th St.

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LXXXIX

RECENT INVESTIGATIONS OF SOUND CONDUCTION

PART I. THE NORMAL EAR

MERLE LAWRENCE, PH.D.

PRINCETON, N. J.

In order for the hair cells to function properly they must be immersed in fluid. If these highly specialized cells were like those of other tissues in the body they could get their energy supply from capillaries, but the very nature of these cells' activity precludes the presence of any structure that might dilate, contract, or pulse. The function of the hair cell is not only to be stimulated mechanically but to be stimulated by forces related only to the form of external physical energy called sound. So of necessity the hair cell is immersed in fluid from which it can absorb nourishment without any extraneous, disturbing influences to make it give off false potentials. In this way the hair cell is very happily situated; it can conveniently carry out its metabolic processes without disturbance, and it sits between two structures, the basilar membrane and the tectorial membrane, so that if the former can be made to move or vibrate a squeezing of the hair cell will result. The big drawback lies in the difficulty of getting the energy of airborne sound into the much more dense fluid which surrounds the hair cell.

The vibrational forces of the air cannot penetrate fluids directly without considerable loss. Acoustically, air is quite a different medium than a liquid and only by means of special devices can energy be transferred from one kind of medium to another with a minimum of energy sacrifice. The middle ear serves such a purpose and a careful study of its achievements in this respect fills one with amazement, for here is a mechanical system that can accomplish what audio-engineers engaged in research on problems of submarine detection for many years have been trying to improve: the transference of vibrations from air to water without most of the energy be-

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ing reflected back at the boundary between the two. What is more, the middle ear accomplishes such a transfer without exerting great resonant peaks of its own into the picture, an achievement microphone manufacturers are still striving for. As if this were not enough the system provides, by means of two muscles, a protective mechanism to prevent overload and subsequent damage of the delicate structures involved.

Experimental studies of the middle ear have all revolved around these three basic functions: (a) the matching of the properties of air to those of perilymph, (b) the transmission of periodic vibrations with a minimum of alteration in their form, and (c) the provision of a protective mechanism for the prevention of damage to the inner-ear structures. An understanding of how these are accomplished is essential to an analysis of auditory loss due to conductive impairment.

THE TRANSFORMER PROPERTIES OF THE MIDDLE EAR

Let us first consider the problem of transferring the energy of vibration from the light particles of air to the heavier ones of liquid. To handle such a problem physicists have formed a concept of the transmittivity of a surface where two mediums meet. This transmittivity is an expression for the ratio between the energy that gets into the fluid to the energy of the air-borne sound oscillating at the surface. A very little calculation shows that only 0.1 of 1% of this energy gets into the fluid, the other 99.9% being reflected back. This means that the intensity of sound in the fluid is 30 db less than that in the air. The inner ear, filled with liquid, should show such a loss of sound energy if the middle ear were removed. It is difficult to measure the energy in the perilymph itself, but the adequacy with which the middle ear matches the air to the fluid can be measured by experiment.

In carrying out animal experiments on the mechanical functions of the sound transmitting systems, electronic recording of the potentials generated within the cochlea serve to indicate, with great sensitivity, changes in transmission properties of anatomical structures prior to the ones which generate the potentials. Generally an electrode of platinum foil is placed on the round window membrane. This electrode leads through a high-gain amplifier to any desired measuring device; a voltmeter, a cathode ray oscilloscope, or a loud

speaker. When care is taken in the preparation of the experimental animal the responses recorded in this manner will remain amazingly constant over long periods of time. In all of the experiments to be described we have used cats because of their convenient size and the easy access to their auditory structures. In order to prevent reflex contractions of the middle ear muscles which would complicate observation the anesthesia was supplemented with curare, which was slowly injected through the femoral vein until respiration ceased. From this point on the animal under deep anesthesia was maintained by a motor-driven artificial respirator, supplementary curare being given from time to time.

In order to get a measure of the animal's hearing for use as a reference level in a manner similar to the 0 db line on the audiogram charts, the level of sound put into the ear of the animal is adjusted to give a certain predetermined amount of response. Measures of the response by means of a voltmeter show it to vary from less than 1/10,000,000 of a volt (0.1 μV) to a maximum of around 1/1000 of a volt (1 mv). This represents a range of greater than 80 db for most of the frequencies studied, from 100 to 10,000 cycles.

By inserting a tube into the external meatus of the experimental animal the amount of sound sufficient to give a response arbitrarily set at 10 μv for 10 different frequencies was determined. If the middle ear is serving adequately the purpose of matching the properties of the air to those of water, the loss of this structure ought to produce a drop close to the 30 db calculated earlier. After removal of all middle ear structures except the footplate of the stapes which had to remain in place for obvious reasons the tube which was formerly in the meatus was moved down over the oval window. It is found that now the same amount of sound as presented to the meatus gives a response much smaller in magnitude. Figure 1 shows an audiogram-like plot of the loss in db due to the removal of the middle ear structure. This loss varies from about 20 to 36 db being most, 30 to 35 db, in the range from 750 to 2000 cycles and again from 5000 to 6000 cycles. The overall average comes out to be about a 28 db loss.[1] The variations in the shape of the curve are very significant and will be considered later.

It is quite obvious that in the normal ear the drum and ossicles simply provide a mechanical advantage so that the light particles of the air can impart their vibrating motions to the heavy particles of

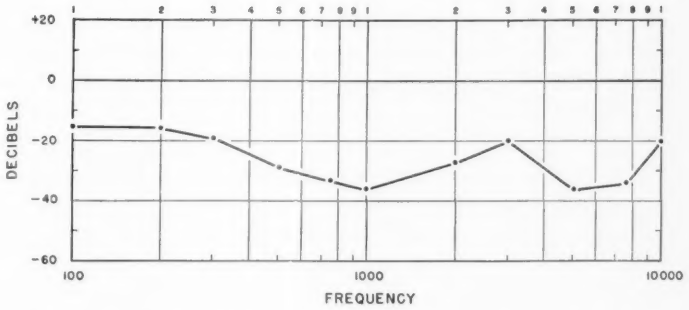

Fig. 1.—The difference in amount of cochlear response when stimulating sound is moved from drum to oval window in the cat. The negative decibels indicate a loss of energy transfer upon removal of the middle ear.

the perilymph. The efficiency of this system in the cat need not, however, be a true picture of that in the human being, but there can be little doubt that the basic function of the middle ear is to serve as a mechanical transformer. The same problem of getting airborne sound into the fluids of the inner ear exists in man and the general form of the ossicular chain is the same so there is little reason to doubt that the human middle ear serves the identical function, and with equal facility, as the middle ear of the cat. Bearing this in mind a great deal of significance can be attached to experiments on these animals.

Several explanations of how the middle ear accomplishes this matching have been proposed. One of these suggested by Helmholtz[2] depends upon the difference in area of the drum membrane and oval window. The force exerted on the larger area of the drum is transmitted through the ossicular chain to the smaller area of the stapes. The transformer ratio would simply be the ratio of the two areas.

Resorting to measurements made on the cat the ratio of the areas of drum membrane to stapes footplate is somewhere in the vicinity of 36.5 to 1. This figure must be reduced somewhat since the effective area will be slightly less than the anatomic area. Consider the drum as a piston surface that in its central region is rigid enough to move as a whole but because of the circular mounting

around the edges this movement tapers off to zero about the circumference. Here we see the significance of the drum's conical shape for such a form adds rigidity to the central portion and enlarges the effective area. Loud speaker diaphragms are built this way for the same reason.

There is no way of knowing what the exact effective area is but certainly at least three-fourths of the total area will be moving in this piston-like manner. The areal ratio now becomes 27.4 to 1. A recalculation of the transmittivity on this basis shows that instead of only 0.1 of 1% getting into the inner ear the amount is 54%. Instead of 30 db being lost in the transfer only 2.6 db does not enter the fluid. There are other factors that make up for this small loss so the transfer is actually quite substantial.

There is very little information on the areal ratio of drum to footplate in man. What there is indicates a variation from a ratio of 28 to 1 to a ratio of 18 to 1. No doubt there is considerable variation among individuals, certainly this is so for the animals reported. From measurements made on the guinea pig the mean ratio is 28.1 to 1. The size of the ratio varies but they are all of the order necessary to provide the increase needed at the footplate of the stapes.

One other hypothesis put forward to explain the ability of the middle ear structure to transmit sound into the perilymph with so little loss has been based on a lever principle. Various estimates have been made as to what this ratio is. Helmholtz estimated 1.5 to 1 which, if such a lever mechanism existed, would add only 1.4 db more gain to that already attained by the difference in area. Such a small amount is difficult to find experimentally because it is in the range of error of measurement. At best it is only a minor method by which the ossicular chain effects the transfer of energy, and there seems to be a good deal of evidence to support the view that the ossicles act as the columella in a bird, remaining rigid as though locked in position, the movements of the stapes footplate being the same as the movements of the drum membrane.

The question might well be asked as to why the ossicles have such an elaborate structure if they function as a columella or stiff rod. A consideration of the finely adjusted protective mechanism of the middle ear may furnish the answer. The tympanic muscles are so situated as to cut down the effectiveness of transmission when sounds of dangerously large amplitudes are present. If the ossicles were

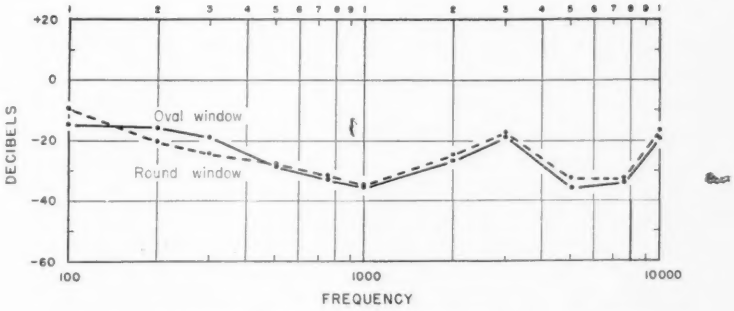

Fig. 2.—The difference in amount of cochlear response when stimulating sound is moved from drum to oval window or to round window of the cat. The negative decibels indicate a loss due to the lack of the middle ear transformer.

actually in the form of a stiff rod the contraction of the muscles could not be anywhere as effective as they actually are in the restriction of movement throughout the middle ear.

It is quite interesting to note that if a sound tube is placed over the round window, the amount of sound required at the different frequencies to give the 10 μV response is within 2 or 3 db of that required at the oval window. Fig. 2 shows a comparison of these two routes. This would indicate that, in the absence of the middle ear, sound energy can enter the fluids of the inner ear just as readily by the round window route as by the oval window though both at a loss of about 30 db. There has always been the question as to whether the round window might not serve as an alternative route for the entry of sound, and from the above evidence there seems to be little doubt of it. DuVerney[3] discussed this problem in 1683, suggesting that the sound vibration could enter the middle ear cavity directly through the drum, passing from there through the round window to the inferior portion of the cochlea. This is no doubt just what happens. As the sound strikes the tympanic membrane setting it in motion, alterations of pressure result in the middle ear cavity. Because of the size of the wave-length of sound with respect to this cavity the effect will be one of increasing, practically simultaneously, the pressure on the inner walls of this space. Such variations of pressure throughout the middle ear space will impinge upon the round

window membrane and be transmitted with a loss of 30 db to the perilymph. It has often been suggested that these vibrations might interfere with the movements of the basilar membrane caused by vibrations of the stapes at the other end of the system.

We have investigated the possibilities of such an effect by measuring the cochlear potentials resulting from the simultaneous presentation of two tones each entering the inner ear by a separate route.[4] The output of an oscillator was divided by a transformer net-work into two separate channels. Each of these channels could be controlled for intensity by attenuators and for phase relations by a phase changer placed in one channel. The terminations were again small tubes with concentric pick-up probes. One of these was tied into the external meatus with the tip as close to the drum as possible, the other was carefully and tightly sealed with wax over the round window. By careful adjustment these two tubes could be so sealed that any appreciable escape of sound was eliminated, all energy being directed at the sealed-in membrane. Generally a platinum foil electrode was in contact with the round window membrane, the fine wire passing out the wax seal. For some experiments, however, a needle electrode was placed at some point along the cochlear wall. In any case the placement or kind of electrode made no difference to the results. An indifferent electrode was always placed in some nearby tissue.

The first step was designed to test the possibility of cancellation or augmentation of the response when sound was sent simultaneously into the inner ear by the two approaches: one by the drum-ossicular route, the other by the round window route. The tone transmitted through the meatus was turned on alone and adjusted in intensity until a response of 10μV was obtained, this was then turned off and the tone to the round window turned on and also adjusted to give a response of 10 μV. The two tones were then turned on together and the response noted. The magnitude of this response under these conditions depends solely on the phase relation of the two tones. At 1000 cycles the oval window and round window make very small vibratory motions, each set of in and out movements taking only 0.001 seconds. To picture the phase relations at the two windows it is necessary to deal with these 0.001 second units, since the resulting response depends upon the time relationship between these units at the two windows. If both are going in at exactly the same moment

they are in phase, and we can call this a zero phase relationship. If one window is going in while the other is coming out we speak of one being 180° out of phase with the other, which means that the in or out movement of one window occurs 0.0005 second after or before the other at 1000 cycles. The differences in phase can of course vary from zero phase to 180° phase, each degree expression representing the time difference in the respective in and out movements. A 45° phase difference at 1000 cycles indicates that the in and out movements of one window follow those of the other with a lag of .000125 second (125 μ second).

In the above experiment it was found that as the phase relation varied, the magnitude of the response changed from double the "one-window" response to almost complete cancellation. No doubt complete cancellation could be obtained with very careful adjustment of the two stimuli. We were limited by having only 1 db steps on the attenuators and 1° steps on the phase changer. The minimum response always occurred at a phase relation 180° from that at which the maximum was observed.

This effect of a 6 db improvement changing to an infinite loss depends very acutely upon two things: the level of response at which the ear is working, and the equality of the two responses arising from the two routes of sound.

A characteristic of the cochlear response is its non-linearity when the stimulating sound becomes quite intense.[5] This probably represents a form of distortion in the response of the maximally responding hair cells. If the two sound sources of the experiment above are raised in intensity so as to give rise to a response near this distortion point, simultaneous presentation of the two sounds in the proper phase relation will not now give a maximum of double the "one-window" response. Indeed, if this is carried far enough even at phase relations which would ordinarily give a "double" response, a loss may occur and damage to the hair cells results. Such an occurrence, however, comes about only under these very special circumstances, and depends upon careful equating of the two responses.

If the intensity of the two sound sources is such that one gives rise to a response much different from that brought about by the other source, exact doubling or complete cancellation will not occur, but the magnitude of the resultant response for certain phase rela-

tions can always be predicted because they are merely the vector sum of the separate responses.

In the normal ear, the vibrations entering by the drum-ossicle route have at least as much as 1000 times the energy of those entering by the round window. Under the most favorable phase conditions the interfering effect of the round window route would be only about 0.3 db. This is negligible. It is only in pathological conditions where there is no workable ossicular chain that the influence of the round window route is a serious problem.

All of the measurements just described do not tell what the exact phase relations are at the two windows or at the involved hair cells when a maximum or minimum response occurs. So far the only thing determined has been that there is a 180° difference in the phase relation between that relation giving a maximum and that giving a minimum. The exact relation was determined by first removing all of the middle ear structures in the animal then sealing tightly over the oval window the tube that was formerly tied into the meatus. The concentric sound pick-up probes were calibrated with respect to each other so that the exact acoustic phase relation at the two windows could be determined. By adjusting the two stimuli so as to give rise to responses of the same magnitude and repeating the procedure followed when the middle ear was present it was found that the minimum response, complete cancellation, occurred when the two sources were in phase, i.e. both attempting to push in and pull out at the same time. The maximum response occurred when one was pulling while the other was pushing and vice versa.

These observations seem to indicate quite clearly that the nature of cochlear action in response to sound vibrations is one of simple mechanical mobility, and that the phase relations of sound entering the two windows merely expresses the amount of mobility permitted in the cochlear fluid. The action is more readily understood when considered as mechanical rather than acoustical, and a few simple considerations should bear this out.

In an average conversation taking place 3 feet from the ear the particles at the eardrum responding to 1000 cycles move back and forth through a total distance of about 0.004 mm (twice the maximum amplitude of 1000 cycles, 60 db above threshold). They

will do this every 0.001 of a second and since this disturbance can move 1,130 feet a second under normal conditions the wave length will be 1.13 feet long. But this is in air. Actually from the time a disturbance impinges on the ear drum the route of travel involves bone and fluid except of course for the air filling the middle ear space which as we have seen need not concern us when considering the normal ear. In sea water, to which we may liken the perilymph, sound travels at 4,950 feet per second, so that now at 1000 cycles a single wave will have exerted its influence over a distance of 5 feet before the next one comes along. This distance is 10 feet at 500 cycles, and 2.5 feet at 2000 cycles, dimensions all so large with respect to those of the ear that it is highly unlikely that the vibrational disturbances are complicated by any phenomena such as eddies or standing waves.

The suggestion has also been made that the middle ear cavity together with the mastoid cells can act as a resonator and that changes in shape or size can alter favorably certain frequencies. The relation between the size of these cavities and the wave length of the frequencies concerned must be kept in mind, for only when the dimensions of the cavity are the same order of magnitude as the wave lengths concerned can resonance occur. Other than that they tend only to complicate mechanical factors of the reacting structures. Thus one would expect the air of the tympanic cavity to add to the stiffness of the drum as it makes its inward and outward movements. We have tried to get an idea of the amount of this effect by recording the cochlear potentials in cats while the bulla was either opened or closed. The difference in sensitivity under these two conditions is not very marked. When the bulla is opened there is some improvement in sensitivity for the tones between 200 and 1000 cycles never amount to more than 5 db. The tones higher than that, up to 10,000 cycles, show little change, if anything they are diminished in sensitivity by about 1 db. We can conclude from this that the actual cavity effects are quite negligible under normal conditions adding only a little stiffness to the operation of the drum.

THE RESONANCE PROPERTIES OF THE MIDDLE EAR

We come now to a very serious problem in considering the general contribution of the middle ear to sensitivity. It is impossible to put a transformer into a vibrating system for the purpose of transferring energy without the transformer itself exerting an influence upon the vibrations. We have just seen that the middle ear

is such a transformer, but we have not yet considerd the other remarkable features of this system other than its ability to effect a power transfer. For this purpose let us consider the drum and ossicular chain as an isolated structure put into forced oscillation by the pushing and pulling of the air particles acting on the drum.

When a vibratory force is first applied to a mechanical system there are actually two different motions set up resulting in a most complicated combination. One of these motions is due to the resonant characteristics of the driven system and the other motion is due to the vibration of the driver. After a very brief interval, however, the first motion drops out of the picture as such and only the response to the second remains. When this stage is reached the motion is described as "the steady state." Although the driven system does not now show any tendency to vibrate at its own natural frequency, those characteristics which determine the natural frequency are still operating and exerting an influence, and under these conditions the driven system is, obviously enough, said to be undergoing "forced" vibration.

A system vibrating in this manner, though doing so at the same frequency as the driver, may differ from the driving frequency in two ways. Firstly, it may respond with a different amplitude. If, for instance, the air particles are doing their pushing and pulling through a certain distance the object upon which these air particles are operating may respond in like manner but through a smaller distance. The degree of amplitude difference is a measure of its sensitivity and will not be the same for all frequencies. Secondly, the forcedly vibrating system may not reach the peak of a push or a pull at the same instant the driver does. As the driver starts a push it may take a brief period of time for the responding system to get under way. At the peak of the push when the driver starts going in the opposite direction the responding system will still be lagging, not reversing its direction for the same brief period. Such a difference in time relation is the same as described earlier and also is called a difference in phase. Any phase shift introduced by the ossicular transformer produces vibrations that are not the same as those in the air. This amounts to a phase distortion and as with the changes in amplitude the phase shift will not be the same for all frequencies.

An analysis of the changes in amplitude and phase at various frequencies introduced by a system undergoing forced vibrations

throws considerable light on the way this system is made up. The middle ear has to be forced into vibration by the driving alternating pressures of the air. Imagine these air-borne vibrations as starting at a low frequency of 100 cycles and then continually increasing up to 10,000 cycles. The ideal system would, of course, respond with equal amplitude and the same phase all along the line, but such a degree of perfection does not exist. The only way audio-engineers come anywhere near accomplishing this in their various recording devices is by means of supplementary electronic networks, boosting one frequency region a little and attenuating another.

If the middle ear behaved like a heavy weight suspended by light spring which was moved up and down at various frequencies the weight-spring system would move with the greatest amplitude at some low frequency. In other words, mass would predominate. Forced vibrations of high frequency would not have time to overcome the inertia of this mass but at a certain low frequency the mass and driving frequency would suit each other and large amplitudes of vibration would result. This would be the natural frequency of such a system. On the other hand if the mass were small and the spring had more stiffness, the natural frequency would be much higher. A violin string is tuned to a high frequency by drawing it tighter or stiffening it, not by adding weight.

Obviously the middle ear mechanism would not be a very successful device if it were tuned to one frequency. If that were so one frequency of speech or music would come through the ear so loudly that nothing else could be distinguished. Ideally the middle ear should respond equally well to all frequencies. In other words the steady state amplitude should be independent of the frequency. There are three ways of doing this but each has its frequency limitations.

If one wishes the system to respond with no resonances in a high frequency region, the system can be given a natural frequency to a low tone by increasing its mass. Thus an oscillator is mass controlled in a frequency range well above the natural frequency. Such a mechanical system would be very insensitive. At its natural frequency the amplitude might exceed that of the driving frequency but the further away from this frequency the greater the loss of sensitivity. Obviously the ear which must respond to a wide range

of tones could not use such a system because the loss in sensitivity would be too great.

If it is desired to have the system respond equally to a low range of frequencies the natural frequency can be tuned very high by increasing the stiffness. Thus an oscillator is stiffness controlled in a frequency range well below the natural frequency. Here again the loss in sensitivity at frequencies other than the natural one would be too great considering the range of frequencies to which the ear responds. A mass or stiffness controlled system is of value only when a small range of frequencies is concerned and where a loss of sensitivity is no great handicap. Designers of phonograph pick up heads and like instruments can use such a method of tuning since any loss of sensitivity can be made up later by the use of electronic amplifiers.

Another way of cutting out resonant peaks in the frequency region of interest and maintaining sensitivity is to increase the friction with which this vibrating system has to work. The smaller the friction is the greater will be the amplitude of movement of the resonator at its natural frequency. By increasing this resistance factor the amplitude at resonance is considerably reduced. The remarkable thing is that the steady-state motion of the resonating system is not very sensitive to changes of this resistance factor except at the natural frequency. When the response of a vibrating system is flattened out in this manner it is said to be resistance controlled.

From these considerations it can be seen that by proper selection of mass, stiffness, and resistance one can, within limits, determine the range over which an oscillator will operate and the manner in which it will behave in this region.

Impairment of the human middle ear system, short of complete removal, can alter these three factors in an unpredictable way, and before such alterations can be understood it is necessary to consider the way mass, stiffness and resistance operate in tuning the middle ear over such a broad frequency range. Again we resort to animal experiments, and in order to have sufficient data to analyse the role played by the three factors both the sensitivity and the phase characteristics of the middle ear must be determined.

We have made such measurements again in cats by making use of the cochlear potentials.[6] Briefly the procedure is to record the

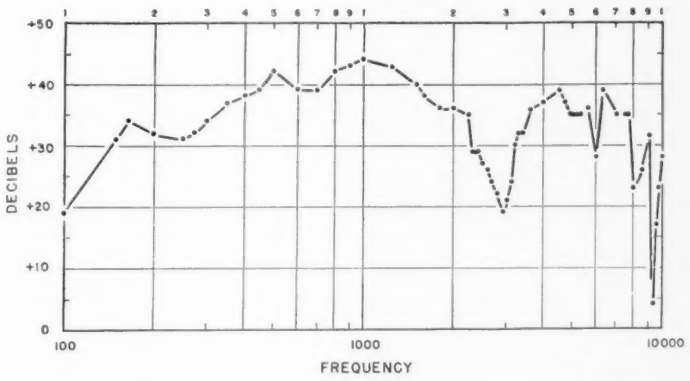

Fig. 3.—The resonance characteristics of the normally functioning drum, malleus and incus of the cat.

amount of sound at the drum necessary to give a response of 10 μV at various frequencies from 100 to 10,000 cycles. This is then compared with the amount of sound at the oval window necessary to give the same response. This is exactly the same sort of measurement made to test the transformer action of the middle ear, but this time the plot is inverted to show the contributions of the ossicular system to sensitivity. In Fig. 3 the abscissa represents the forcing frequency applied to the middle ear, while the ordinate shows the magnitude of response in dbs relative to the cochlear response with no middle ear present. The forced motion is shown in dbs rather than in a non-logarithmic plot because we are interested in the way in which middle ear resonances may be reflected in hearing-loss audiograms. Several features of this curve are important, keeping in mind that this is the ear of a specific cat. There appear to be two regions of maximum sensitivity, one at 1000 cycles and another centered in the vicinity of 4500 to 6300 cycles. Between these two regions, at 2900 cycles, there is a point of extreme insensitivity. Such a curve is quite common in the cat, appearing in the majority of those studied, and probably represents a double-tuned system, having two resonant peaks and an anti-resonance in between. The middle ear obviously does not perform its transformer duties without exerting some of its own characteristics, but even at that the system does a remarkable job considering this is a response to

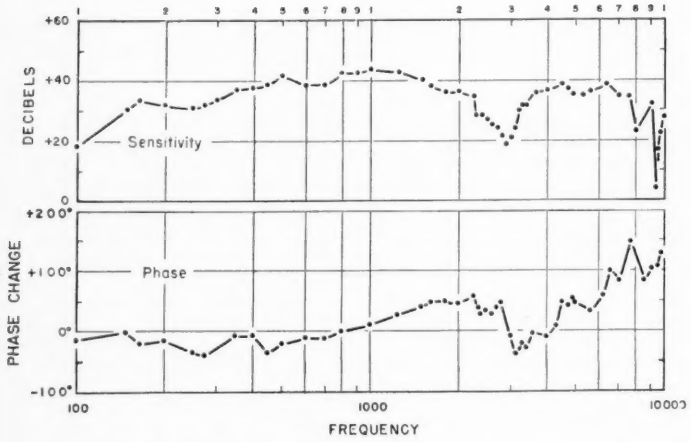

Fig. 4.—The phase changes compared with the resonance characteristics of the normally functioning drum, malleus and incus of the cat.

nearly seven octaves. Apparently there is considerable friction or damping present, in order for the system to be as sensitive as it is and still be responsive over such a range. There are several masses and stiffnesses involved to give such a picture and the anatomical situation certainly provides for the high degree of resistance. No doubt the irregularities of the curve are real and represent individual characteristics of this particular mechanical arrangement.

As already mentioned the phase of the system will also vary as the driving frequency is changed, and these phase changes have a specific relation to the resonant characteristics. Where there are several masses and stiffnesses involved the variations of the phase of displacement with that of the force become quite complicated, but a study of this phase picture throws some light on the resonant characteristic of the responding system. Fig. 4 shows the phase changes introduced by the middle ear as the frequency is varied from 100 to 10,000 cycles. The curve of Fig. 3 is also presented here for comparison with the phase changes. Over the lower range the middle ear produces a small advancement of phase. At about 1000 cycles the displacement is in phase but for frequencies above this up to about 2500 cycles there is an increasing lag behind the driving force. At

3000 cycles they are again in phase; between 3000 and 4000 cycles the middle ear is a little advanced and at 4000 cycles they are again in phase. Above 4000 cycles there is a continued tendency for the phase to lag.

Because of the impracticability of removing the stapes these measurements are for all the middle ear structures peripheral to it. Similar phase measurements were made for the stapes and it was found that it does not complicate the picture until a driving frequency above 6000 cycles is reached.

The significance of these phase findings lies in that they confirm the double tuning characteristic revealed by the sensitivity measurements. In complicated transmission systems the direction of change of phase with frequency indicates the way in which the various masses and stiffnesses are behaving under the vibrations. In general, when, as the driving frequency is raised, a change in the forced phase from an advancement to a lagging takes place the presence of a resonant point is indicated. At this point there should be a region of maximum amplitude. If the phase changes from a lagging one to an advanced one, a point of anti-resonance is indicated and there should be a region of minimum amplitude. With this in mind it can be seen from Fig. 4 that the two resonance points are at 1000 and 4000 cycles corresponding with the two points of maximum amplitude. An anti-resonance point occurs at 3000 cycles and this is the point of minimum amplitude.

Any slight change in mass, stiffness, or damping will alter the picture just described. The curves presented are for a certain cat and vary from cat to cat. In man the middle ear resonance is different; the curve seems to have only one region of maximum sensitivity with considerable less sensitivity for the extreme lows and the highs. This, no doubt, reflects evolutionary changes and the use to which the ear is to be put.

SUMMARY

As it turns out the middle ear has the function of matching the impedance of the air to that of the cochlear fluids. If no such transformer system were present there would be a loss of 30 db in the transfer of energy from air to liquid. The drum and ossicle arrangement manages to overcome this to the extent of 28 db, and does so probably by the simple method of increasing the pressure

at the footplate of the stapes through a concentration of force. This concentration comes about through the large forces acting on the drum being transferred through a chain of bones acting like a columella on the relatively small footplate of the stapes. Even more remarkable is the fact that the middle ear achieves this task while exerting a minimum of its own resonant properties into the picture. Its natural frequency lies near the middle of the audible range, a factor which provides for maximum sensitivity in this region. This system is highly damped to make the response uniform with frequency without sacrificing too much of the sensitivity.

The action of the middle ear on the inner ear fluids is mechanical, requiring that the other end of the system be free to move. The hair cells cannot be stimulated by the presence of sound pressure alone; there must be mobility of the fluids such that the mechanical effects of sound can actually move the basilar membrane back and forth. Sounds of equal energy entering at the two ends of the cochlear system can either double or completely cancel the movement of the hair cells depending upon the phase relation of the two stimulating sounds.

Alterations in the conductive system varying from the slightest changes in mass, stiffness, or friction to complete fixation or removal can produce an unlimited variety of audiometric pictures. The nature of the most common of these I leave to Dr. Wever.

PRINCETON UNIVERSITY.

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XC

RECENT INVESTIGATIONS OF SOUND CONDUCTION

PART II. THE EAR WITH CONDUCTIVE IMPAIRMENT

ERNEST GLEN WEVER, PH.D.

PRINCETON, N. J.

The subject before us, which is the reception and transmission of aerial sounds by the ear, is difficult enough when we deal only with normal conditions, as shown in the foregoing treatment by Dr. Lawrence (Part I under this title). Obviously it grows still more difficult when we add the variations of function that come with pathological changes of the structure. Fortunately, however, we can expect to find in operation in the abnormal ear many of the same general principles that have been disclosed in the normal ear, though oftentimes these principles manifest themselves in somewhat different settings and in company with different variables. The following discussion will deal with some of the common changes of the ear's conductive structure that result from accident or disease or deliberate surgical effort, with the object first of all of developing a clearer formulation of the problems and then of suggesting how the above principles and others may apply to their solution.

1. *Perforation and loss of the drum membrane.* One of the simplest of conductive lesions is a perforation of the drum membrane. It occurs with great frequency in otitis media either as an external rupture under the force of accumulated pus and fluid or as an incision made deliberately in the treatment of the disorder. Ruptures also result accidentally from external objects and large changes of air pressure. Auditory impairments are observed under all these circumstances, but unfortunately it is not a simple matter to ascertain the degree to which they are the result of damage to the drum membrane, for there is always the possibility that the accident or disease has affected other parts of the ear as well. Also, the normal acuity is seldom known, and though a comparison may

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sometimes be made with the condition after healing has taken place, we can never be certain that recovery has been complete.

Animal experiments avoid these difficulties, but of course they include others, and inevitably they bring in whatever peculiar characteristics the chosen species may have. Bordley and Hardy[1] reported results obtained on the cat by measuring the electrical potentials of the cochlea before and after simple incisions were made at different places in the tympanic membrane. They found losses generally of the order of 10 to 20 db. The losses were somewhat greater for incisions in the superior and posterior regions of the membrane and for ones that were carried in an arc partway around the manubrium of the malleus.

The impairments resulting from perforations of the drum membrane vary according to their size and locus because this membrane has several related functions in the process of sound transmission.

(a) Primarily, of course, this membrane is the surface on which sound waves exert their pressure, and is the means by which the total force sets up movements of the ossicular chain. A perforation of the membrane causes a direct reduction in this force, a reduction that is proportional to the area lost.

(b) There is also an indirect effect. A perforation permits the sound to pass through the membrane and to exert pressure on its inner surface, and so to act in opposition to the force on the outer surface. This negative force will be proportional to the area of the perforation.

(c) Because the membrane communicates its motions to the ossicular chain by way of the attachment of the manubrium, a perforation is particularly serious when it is in the vicinity of this attachment.

(d) The drum membrane also is a part of the tuned mechanical system of the ear. It supplies a large element of stiffness to this system. Any perforation of the membrane reduces this stiffness and thereby alters the vibratory characteristics of the system as a whole. This change will be most serious for the higher tones: those above about 1000 cycles.

2. *Interruption of the ossicular chain.* A second form of conductive impairment consists of an interruption or loss of the ossicular chain. It is encountered in clinical experience as a result of otitis

media, accidental injury, and certain operations such as a radical mastoid operation.

Animal experiments give detailed information on the nature of this impairment. Tests carried out on cats show a profound loss of response as the result of a simple interruption of the ossicular chain at the incudostapedial joint, when the parts of the middle ear mechanism are not otherwise disturbed.[2] This loss is of the order of 60 db for tones of the middle range, and somewhat less for the low and high tones. The loss is not as great if in addition the drum membrane and the two outer ossicles are removed. The difference is of the order of 15 to 20 db in the middle range and a little less for the extreme tones. It is evident that when the ossicular chain is interrupted at the stapes the more peripheral parts are worse than useless. They stand in the path of the sound waves and form a partial barrier to their entrance into the tympanic cavity.

The impairment of about 40 db that remains after removal of the drum membrane and the two outer ossicles is the combined result of three conditions.

(a) The principal condition is the loss of the transformer action of the middle ear mechanism. As we have seen (Part I) this transformer action normally improves the transmission of aerial sounds to the cochlea by about 28 db.

(b) A second condition is the modification of cavity effects. When the ear is normal the tympanic air space adds stiffness to the moving parts, because as the drum membrane moves it alternately compresses and rarefies the entrapped tympanic air. However, the stiffness contributed in this way is small in relation to the stiffness of the system as a whole as produced by the drum membrane, the suspensory ligaments, and the fluid of the cochlea. This fact is shown in the cat by opening the auditory bulla and relieving this aerial loading. The difference in sensitivity that results is only slight, and it appears that in the normal condition the elastic reaction of the tympanic cavity aids the reception of high tones to the extent of 1 to 2 db and hinders the reception of low tones to the extent of 2 to 5 db.

(c) A third condition, and one of importance, is the simultaneous entrance of sound to the cochlea by both oval and round windows. To it we can credit a loss of about 12 db.

Under the conditions described, when the drum membrane and outer ossicles are absent, the sound that is applied to the ear has ready access to both windows. Also, the distance between the two windows is so short in relation to the wave lengths of all except the most extreme high tones that the sound pressures exerted at the two locations are practically equal in both magnitude and phase.[3]

Now we know, from tests in which the sounds are applied separately to these windows, that they are almost equally effective by the two routes, with only a little difference in favor of the oval window route. This difference is very likely due to the presence of the stapes in the oval window.[4]

When a sound enters the cochlea by both these routes simultaneously, and with the proper intensity and phase relations, the result is a cancellation of effect.[5] The cancellation occurs because a positive pressure through one window that tends to displace the basilar membrane in one direction is opposed by a positive pressure through the other window that tends to displace this membrane in the opposite direction. No motion of the membrane then occurs.

Fortunately, however, the conditions for complete cancellation are highly critical. If the intensities and phases of the sounds along the two routes are not exactly equal the cancellation is only partial. The 12 db loss shown in this situation represents such a partial cancellation. It is easy to estimate the departures from equality in the sounds along the two routes that give this amount of cancellation. We can assume that the sounds in the two routes have the same phase but differ in amplitude by 2 db, or that they have the same amplitude but differ in phase by 14.5°, or that they differ by smaller amounts in both amplitude and phase.

As just mentioned, the presence of the stapes in the oval window produces small variations in the amplitude of the sound transmitted along its pathway. Further evidence shows that the stapes produces variations of phase also. These changes of phase are small, of the order of a few degrees, over most of the auditory range, but they are important nevertheless. Together with the slight amplitude variations they limit to something like 12 db the cancellation effect that otherwise might carry the sensitivity down to zero.

It is evident that after an interruption of the ossicular chain the sensitivity of the ear would be improved if somehow the sound were prevented from reaching both windows with equal facility.

When the ear is normal the cancellation effect with which we have been dealing is unimportant because the transformer action increases the efficiency of transmission by way of the oval window to the point where the sound entering the round window is wholly negligible. If the sound in the one pathway is 28 db greater than in the other the cancellation effect cannot exceed 0.3 db even with exact phase agreement in the two pathways.

3. *Fixation of the ossicular chain.* A third type of conductive impairmant consists of a fixation of the ossicular mechanism. At least three forms are recognized.

(a) *Fixation by fluid.* One form of fixation appears in the acute stage of middle ear infection from the accumulation of fluid and semifluid substance in the tympanic cavity. When it comes in contact with the drum membrane and ossicles this substance dampens and loads their movements, and when finally it fills the cavity it adds stiffness as well. The result is an impairment of sensitivity that in the early stage affects the high tones the most seriously, and then in the severe stages includes all tones.

(b) *Simple mechanical fixation.* When the acute stage of middle ear infection is over it often leaves behind various modifications of the moving mechanism. Particularly frequent are thickening and scarring of the drum membrane and fibrous adhesions between parts of the moving mechanism or between this mechanism and the adjacent walls of the tympanic cavity.

As Polvogt pointed out,[6] some of these modifications appear in the ears of persons whose hearing had been regarded as normal. He described, for example, an ear in which the drum membrane was adherent to the surface of the promontory. Yet the person when tested a short time before death had shown normal acuity. Polvogt noted also in normal ears many fibrous connections between the stapes and the bony walls near by, and fibrous strands in the round window niche, often attached to the round window membrane.

In a further study, Polvogt and Bordley[7] observed that persons whose hearing was impaired for air-conducted sounds differed from those with normal hearing in that they always had lesions involving one or more of the ossicles, and concluded that such lesions interfere most seriously with the movements set up by sounds.

I think it would be a mistake to say that the pathological changes observed in the ears tested as normal had no effects at all upon the hearing. Our experience in working with the ears of animals, in which we have the cochlear potentials to indicate precisely the changes of transmission, throw doubt on this position. In the animal experiments we find difficulty in making any alteration at all in the conductive mechanism without an immediate modification of the sensitivity pattern. What seems to be an inconsistency here is only a reflection of the crude character of an observer's judgments of intensity changes. Under good conditions an observer can just about detect a 40 per cent change in the amplitude of a sound, which in logarithmic terms is about 3 db. Under clinical conditions we cannot trust the observations more closely than 5 to 10 db. An impairment of the conductive mechanism that reduces its transmission to one-half the normal value, which is a 6 db loss on the logarithmic scale, may easily go unnoticed. It is indeed fortunate that we can make this physical sacrifice without too great a perceptual loss. It is when a number of these physical changes occur together in the same ear that the impairment becomes serious. Six or ten lesions, if each contributes a loss of 5 to 6 db, add up to a crippling total. Such multiple lesions appear to be the rule in pathological ears. Polvogt and Bordley found that the great majority of impaired ears had changes in every one of the 10 structural areas into which they had divided the middle ear for study. It must be further emphasized that certain lesions, especially those involving the ossicular chain, may give losses far exceeding 5 or 6 db.

Some of the animal experiments demonstrate how serious a simple mechanical fixation of the ossicles may be. In a series of experiments on cats reported by Smith,[8] a steel needle was anchored in the auditory bulla so that its midportion passed close by the head of the stapes, and the stapes then was tied firmly to the needle by a thread. As a result of this procedure the sound transmission was reduced in different trials from 15 to 55 db, depending on the security of the anchorage. A reduction of 55 db represents a physical fixation of 99.8 per cent: the amplitude of the conducted sound was dropped to 0.2 of 1 per cent of its normal value. The average results obtained in this experiment are shown in Fig. 1. It is clear that the impairment produced by this form of fixation is related to frequency, as the losses of response are progressively greater for the lower tones. This effect is the one to be expected if the fixation adds

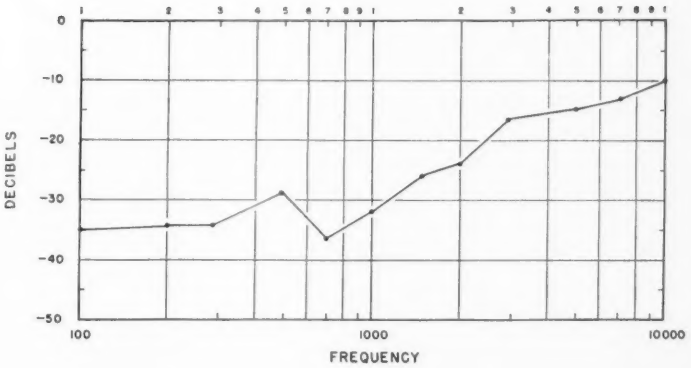

Fig. 1.—The effects of a mechanical fixation of the ossicular chain on air conduction. The observations were made on the cat by Smith.[8]

a large amount of stiffness as well as frictional resistance to the moving system.

(c) *Fixation by otosclerosis.* The most impressive type of fixation of the ossicular mechanism, and the one of greatest clinical importance, occurs in the disease of otosclerosis. As is well known, this fixation results from an invasion of the oval window by bone tissue and a final ankylosis of the stapes footplate to the cochlear capsule. Its clinical result is a progressive impairment of hearing, usually extending over a number of years, to an amount that may exceed 80 db.

It is generally agreed that in the early stage of this disease the impairment is progressively more serious for the low tones, as shown in Fig. 2. This result corresponds to what has been shown for a mechanical fixation in the cat, and evidently means that the invading bone has added greatly to the stiffness of the moving system. Later, as the fixation continues, the middle and high tones are affected further, and still later, in many examples at least, the high tones drop out altogether.

It is a question whether this high-tone loss is a continuation of the effects of the otosclerotic process by which the low-tone loss arises, or is an independent process that sometimes is added to the

other. It is possible that both these explanations are valid in different instances. A high-tone deafness like that ordinarily regarded as old-age deafness, and due to degenerative changes in the sensory cells or the cochlear nerve fibers, may easily occur in an otosclerotic patient quite independently of that disease. Or, such degenerative changes may be a secondary effect of disease, arising from toxic products of the lesion, but still may be independent of the mechanical fixation process. It is also possible to explain the high-tone loss as a result of the fixation. As will be brought out presently, the immobilizing of the cochlear fluid gives an impairment of all tones.

THE FENESTRATION OPERATION

One of the great discoveries of otology is that the clinical symptoms of otosclerosis are relieved by fenestration of the labyrinth. Even when the sensitivity has fallen 60 or 80 db or more below normal it makes a dramatic return when an opening is made through the bone to the perilymphatic space at the vestibular end of the labyrinth.

Several kinds of fenestrations are possible, and all are beneficial in some degree as long as they satisfy the conditions just mentioned. Within the limits of surgical access, the opening may be made in any one of the semicircular canals, and anywhere in its course, or in the vestibule proper; and in every case—barring accidents—the improvement follows.[10] There may even be two or more openings. The surgical approach may vary, and the middle ear structures may be variously handled. Thus Holmgren approached the labyrinth by the post-auricular route and left the middle ear mechanism undisturbed, whereas Lempert[11] in the modern operation employs the endaural approach and greatly modifies the middle ear mechanism by displacing the drum membrane in position and removing the incus and part of the malleus and sometimes the crura of the stapes as well. Both procedures produce desirable results, though, to be sure, not equal results. On the other hand, an opening into the scala tympani will not serve.

The improvement of hearing that follows this surgical treatment is diminished and lost if the fenestra is closed by fibrous and bony growth. It is regained when the fenestra is then reopened.

From all these observations we are assured that the important thing in the improvement of hearing by fenestration is that there

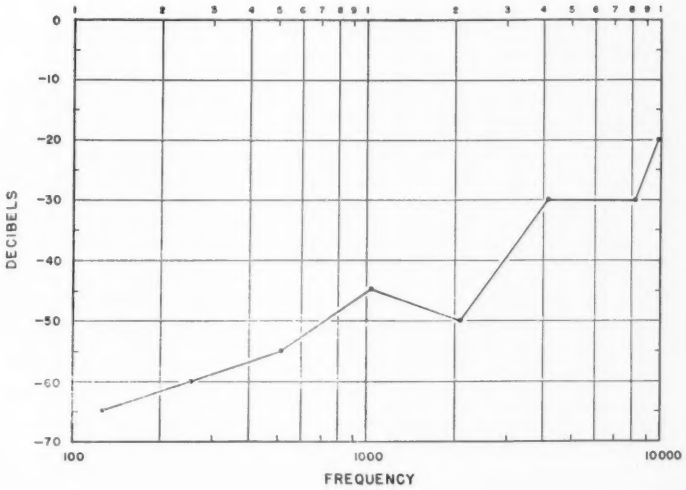

Fig. 2.—Otosclerosis at its early stage. From Lempert.[9]

shall be an opening of the perilymphatic space at the vestibular end of the labyrinth. We now have to explain how this opening aids in the process of hearing.

In this explanation three theories have been advanced: the decompression theory, Sourdille's 'new tympanic system' theory, and the mobilization theory. The decompression theory, held by Holmgren, asserts that the fenestration restores hearing by relieving an excess of perilymphatic fluid pressure. As has already been pointed out,[12] this theory suffers from several basic weaknesses. It has never been demonstrated that an excess of fluid is present in otosclerosis, and, besides, it can be shown that a raising of the fluid pressure does not impair the cochlear action.

Sourdille's theory is that the tympanomeatal flap in association with the incus (which he preserves) acts as "an amplifier of sonorous vibrations."[13] He conceives that the vibrations travel to the fenestra along the skin flap. However, this idea encounters a difficulty in that by the process of healing this flap becomes firmly adherent to the underlying bone and then is no longer free to move. Also, as

Dr. Lempert has observed, the flap may be completely severed between drum membrane and fenestra without any noticeable effect upon the hearing.

The mobilization theory remains. This theory was suggested by Lempert[14] in his presentation in 1938 of the one-stage fenestration operation and was more explicitly stated in his further reports of 1940 and 1941. He conceived that the opening of the labyrinth permits air-borne sound more effectively to set up a motion of the perilymphatic and endolymphatic fluids. The new opening in a sense takes the place of the occluded oval window and once more permits the particular kind of pressure discharge between the scala vestibuli and the scala tympani that involves the basilar membrane and stimulates its sensory cells.

This theory is consistent with all the observations so far mentioned. It explains why the fenestra must be in the vestibular part of the labyrinth and not in the tympanic part. For a pressure discharge to traverse the basilar membrane there must be two openings, one in the tympanic region which is provided in the round window and another in the vestibular region which is surgically created.

This theory also explains why, as is generally reported, the hearing is best at the moment that the new opening is made, and recedes somewhat when this opening is covered with a skin flap. It explains likewise the progressive loss as the fenestra is invaded by fibrous tissue and finally is closed by bony growth, and it explains the return when this closing tissue is removed in a revision operation.

Further observations made on experimental animals also are consistent with this theory. Let me mention first some experiments carried out on monkeys by Drs. Lempert, Meltzer, Lawrence, and me. The cochlear potentials were observed by means of an electrode on the round window membrane, and were measured first after simple exposure and then after three further procedures were carried out. These procedures consisted of breaking the ossicular chain, then closing the oval window and fixing the stapes by the application of dental cement, and finally fenestration of the lateral semicircular canal. The breaking of the ossicular chain, as expected, seriously reduced the responses. The closure and fixation of the oval window reduced them further, and by so much as to make them no longer measurable. The fenestration returned the responses to about the level before the fix-

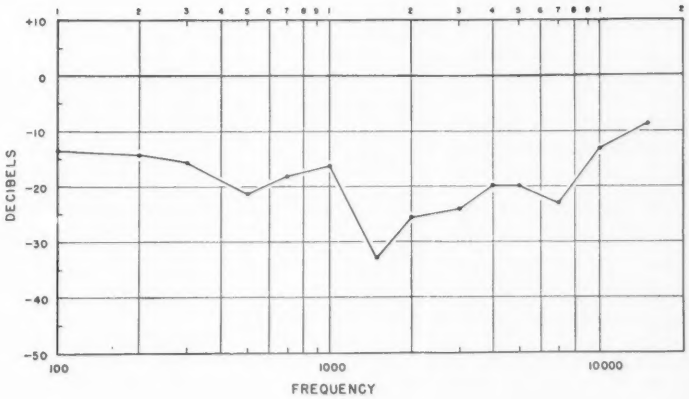

Fig. 3.—The effects of blocking the oval window. Cat No. 838R.

ation. Though these tests do not give quantitative indications of the benefits of fenestration (because the fixation depressed the responses too far) they do provide objective evidence of them.

Dr. Lawrence and I have carried out further experiments on the cat, also by use of the cochlear potentials. In one series we first broke the ossicular chain at the incudostapedial joint and removed the drum membrane, malleus, and incus. Then we stimulated with sounds introduced through a tube carefully sealed over the round window. The sensitivity by this route is very nearly the same as if (with the peripheral part of the conductive mechanism likewise absent) we introduce the sound through a tube over the oval window, as already mentioned. Then we produced a fixation of the oval window by packing with wax. For this purpose we used surgical bone wax and applied it with great care, yet thoroughly, so as to embed the stapes fully and to build up a considerable mass about it. The result of this fixation, as in the monkey experiments, was a marked reduction of responses. In this animal the potentials were still measurable, as Fig. 3 shows. The impairment varied with frequency, but amounted on the average to about 20 db. When the wax was removed as completely as possible the responses returned to almost their former level. We have used the wax for this fixation in preference to cements,

though no doubt these would give greater reductions, because the wax is largely removable and allows us to prove that there has not been any accidental damage to the cochlea.

A number of persons, including ourselves, have carried out the counterpart of this experiment, which is the blocking of the round window while stimulating by way of the oval window.[15] The results of these experiments have been surprising because they have revealed only slight changes of conduction as a result of the blocking. For the most part—with the exception of Hughson's experiments, which I think can be set aside—these results show impairments, but impairments that are slight in relation to what we expect on the basis of the principles that we are now considering.

We have recently returned to this experiment, and we think we now have the explanation of the divergency. It arises from the extreme difficulty of producing a true block of the round window and the slight magnitude of the relief that the cochlear fluid requires for effective stimulation. Ordinary plugs that are inserted into the round window niche simply vibrate bodily with the membrane. By their presence they modify the response function a bit but they do not immobilize the fluid. Even a heavy layer of substance over the window is of slight effectiveness if, as may happen, it entraps a bit of air next to the membrane, for then a part of the membrane can move in and out of this air bubble. The minute amount of air required to permit this movement will be appreciated if we calculate the volume displacement of the cochlear fluid for some representative sound. For 1000 cycles at a moderately strong intensity (1 dyne per sq cm) this volume displacement is 3 ten-millionths of a cubic millimeter—the volume of a sphere of about the diameter of a red blood corpuscle. An air bubble need be only a million times this size—about one-third of a cubic millimeter—to be reasonably effective in mobilizing this fluid.

We have found it possible, by the use of extraordinary care, to block this window adequately with wax and to obtain impairments practically as great as those obtained at the oval window. Fig. 4 gives some of the results, for two degrees of blocking. These effects disappear and the ear returns to normal when the block is removed.

A PHYSICAL THEORY OF COCHLEAR IMMOBILIZATION

We now require a physical explanation of the immobilizing effect of a blocking of one or the other of the cochlear windows.

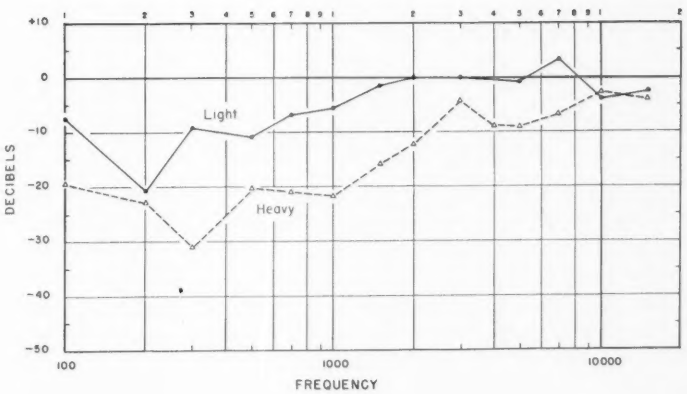

Fig. 4.—The effects of blocking the round window. Cat No. 840R.

This explanation may first be sought in a consideration of impedance conditions. Let us examine these conditions in advanced otosclerosis when only the round window is functional. Sound waves acting upon the round window membrane encounter great resistance because this membrane reflects the characteristics of the bony capsule. We can calculate a maximum value for the resulting impedance. If the characteristics of the bony capsule are fully reflected upon the membrane (which is equivalent to saying that the cochlear contents have been converted to solid bone) we find that only 0.03 of 1 percent of the energy of the incident sound wave will enter the cochlea; the remaining 99.97 percent is reflected from the membrane surface. In logarithmic terms this loss amounts to 35 db, and is only 5 db more than the loss sustained when aerial sound enters an unbounded fluid.

This amount of loss is clearly inadequate to explain the great impairments of sensitivity that occur in otosclerosis. They are likewise inadequate to explain the results of our blocking experiments, where the total reductions, adding on that due to the absence of the middle ear mechanism, amount to as much as 60 db.

The cue to a solution of this problem comes in a further consideration of the stimulation process in the cochlea. For a stimula-

tion of the sensory cells it is not enough that sound energy be present in the cochlea; this energy must be channeled across the basilar membrane in such a way as to set up movements of the membrane. We have experimental evidence for the validity of this principle. It was obtained in cats by the measurement of cochlear potentials on the presentation of sounds simultaneously to oval and round windows.[5]

When a given sound is presented to these windows along separate channels at the same effective intensity, and the phase relation of the waves in the two channels is altered, we find that the resulting responses vary in magnitude all the way from twice that of a single channel to zero. The doubling of response occurs when the waves that are passing through the two windows are in phase opposition, for such waves on reaching the basilar membrane are operating in contrary directions and hence in an effective sense are in phase agreement. The membrane is being pushed from one side and at the same time pulled from the other side. Contrariwise, the cancellation of response occurs when the waves near the two windows are in phase agreement, for then at the basilar membrane they are effectively in phase opposition. The membrane then is being pushed (or pulled) from both sides simultaneously.

The maximum pressure is exerted upon the hair cells when the waves at the basilar membrane are in phase opposition, yet these cells then remain unstimulated. The maximum displacement of the basilar membrane occurs when the waves at the membrane are in phase agreement, and this is the condition under which the hair cells are most effectively stimulated.

When the cochlear capsule contains only one opening and sound energy enters through this opening the energy spreads throughout the capsule. Because the dimensions are small relative to the velocity of propagation of the energy the spread for all practical purposes is instantaneous and the pressure at any given moment is the same at all points within the capsule. This energy is dissipated in all directions; it is used up in the expansion of the capsule or the displacement of the fluid in and out of connecting blood vessels and nerve channels. There is no well-defined channeling of it with respect to the basilar membrane and therefore there is little or no movement of the basilar membrane. This condition is portrayed in Fig. 5.

When, on the other hand, there is a second opening in the capsule in a part of the wall that lies across the basilar membrane from

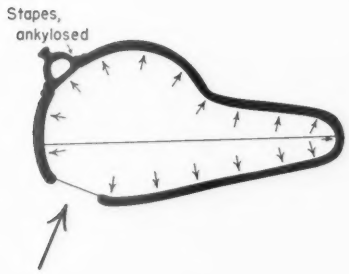

Fig. 5.—Aerial stimulation in the otosclerotic ear. The cochlea is presented in schematic form, with upper and lower chambers separated by the basilar membrane. The upper chamber is a combination of scala vestibuli, cochlear duct, and the free space of the vestibule; the lower chamber is the scala tympani. The stapes is represented as ankylosed, and aerial sound, as indicated by the large arrow, is applied at the round window. Its pressure is distributed uniformly over the inner wall of the cochlear capsule, as the small arrows show. At any point within the capsule, including all points along the basilar membrane, the pressures are equal in all directions, and therefore the basilar membrane is not displaced.

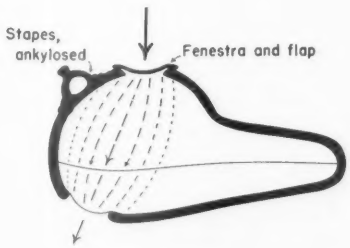

Fig. 6.—Aerial stimulation in the fenestrated ear. The cochlea is represented as in the foregoing figure except that now a fenestra, covered over with a flexible skin flap, is present in the vestibular region. Sound applied at the fenestra is channeled across the cochlea to the round window. The large arrow indicates that at a given instant a positive pressure is exerted at the fenestra, and the small arrows suggest the direction of the pressure discharge whereby the basilar membrane is pushed downward. A little later, as the vibration reverses its direction, all these pressures will be reversed and the basilar membrane will be pulled upward. The stimulation would be equally effective if the sound were applied at the round window.

the first opening, the action changes. The impedance becomes less, but this is not the main thing. The main thing is that now the sound pressure exerted on one opening displaces a quantity of fluid leading to the other opening. It vibrates this fluid and moves the basilar membrane in doing so, and thereby stimulates its sensory cells. Fig. 6 presents this situation.

This mobilization effect operates similarly for all audible frequencies, because all have wave lengths in the fluid that are large with respect to the dimensions of the cochlear cavity.

ACOUSTIC ROUTES TO THE FENESTRATED EAR

Still to be considered is the problem of the pathway of entrance of sound to the cochlea after the fenestration operation. It has sometimes been suggested that this pathway is the new fenestra and at other times that it is the round window. Let us first examine the physical situation.

In the Lempert operation as performed at the present time there is a surgical reconstruction of the tympanic structure, which is designed primarily to provide an epithelial covering of the fenestra, a provision that is essential to the continued preservation of the membranous labyrinth. The drum membrane except at its inferior and anterior margin is freed from its bony sulcus. This membrane together with a portion of the cutaneous wall of the meatus is then displaced so that its meatal portion covers the fenestra. This displacement brings the tympanic portion of this tympanomeatal membrane over a part of the tympanic space that is now smaller than the original tympanic cavity. This reduced cavity contains the round window.

A larger cavity remains as an extension of the meatus. It includes a small part of the original tympanic cavity, the epitympanic space from which the incus and the body of the malleus have been extracted, and a considerably enlarged mastoid space. In this larger cavity lies the fenestra with its transplanted covering.

Sounds have an unobstructed path from the outside to this cavity containing the fenestra. They may reach the round window after traversing the drum membrane. Two series of observations bear on the question of the relative effectiveness of these two routes.

(a) Dr. Lempert has observed that if at any time during the operation or afterwards a perforation is made in the drum membrane

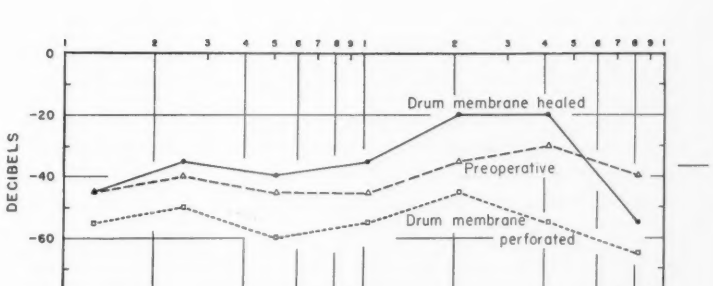

Fig. 7.—The effects of a perforation of the drum membrane in the fenestrated ear. In this ear the drum membrane was accidentally torn in the course of the operation. The lowermost curve shows the acuity 4 weeks after the operation, at a time when ordinarily the hearing would have been improved. Instead, the hearing was worse than it had been preoperatively. Still later, 10 weeks after the operation, when the perforation in the drum membrane had become healed, the hearing was greatly improved, especially for the medium high frequencies. From Dr. Lempert's case records.

the hearing is immediately impaired. When later the perforation heals the hearing is restored to the level obtained before this injury. Fig. 7 shows some results of this kind.

(b) Dr. Lempert has also performed the following test on fenestrated ears after complete healing has taken place and a satisfactory recovery of hearing has been obtained. A quantity of vaseline cotton was packed upon and around the tympanic membrane with view to sealing this membrane from the exterior as thoroughly as possible and preventing the passage of sound waves through it to the round window. The path to the fenestra remained unobstructed. Audiometer tests carried out before and after the packing and again when the packing was removed showed only a slight effect upon sensitivity. Sample results are given in Fig. 8. As will be seen, most of the tones showed reductions of 5 db as a result of the packing.

The observations on the effects of perforations in the drum membrane show that the round window is involved in the transmission of sounds to the fenestrated ear, whereas the observations

on the effects of packing the drum membrane show that the round window is not the only route. Evidently sounds enter the cochlea simultaneously by both routes: by way of the fenestra and by way of the round window.

It is further evident that the sound does not enter by these two routes equally and in the same phase. The animal studies, as already described, show that when this happens there is a cancellation of effect. The absence of such a cancellation means that there are changes of intensity or phase, or both, in the fenestral and round window routes to the cochlea.

A reasonable hypothesis is that these changes are mainly due to the presence of the drum membrane in the round window pathway. When this membrane is perforated its phase-changing action is impaired and a partial cancellation of the sound action is the result. When later the perforation heals the phase changing action is restored, and the ear's response is improved.

Our studies on the cat lend credibility to this assumption of phase changing action by the drum membrane.[16] We have observed such action as carried out by the drum membrane in combination with the malleus and incus, and a similar performance is to be expected of the membrane and part of the malleus only.

It appears likely that the phase change produced by the drum membrane, for some tones at least, exceeds 90° and even approaches 180°, in view of the observation that when the round window pathway is blocked off the ear's acuity is impaired. If the impairment were 6 db we should conclude that the phase difference had been 180° and that the two windows had been operating equally in conveying the sound to the basilar membrane, for then the blocking of one pathway would cut the conducted sound in half.

Smaller impairments signify that the reinforcement falls short of the maximum value, either because the phase difference does not attain 180° or because the intensities in the two pathways are not equal, or for both these reasons.

BONE CONDUCTION IN THE OTOSCLEROTIC EAR

A further question concerns bone conduction in the otosclerotic ear before and after the fenestration operation. The general opinion is that bone-conduction sensitivity changes little if at all as

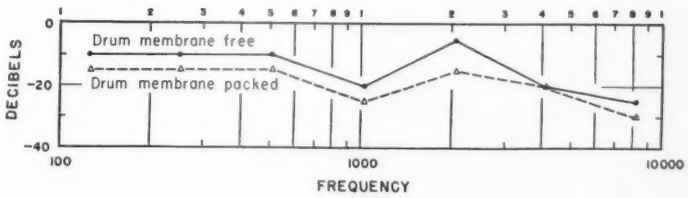

Fig. 8.—The effects of blocking the drum membrane in a fenestrated ear. The upper curve shows the acuity in advance of the packing procedure, and the lower curve shows the acuity somewhat depressed as a result of a blocking of the drum membrane. The acuity reurned to its former level when the packing was removed. From observations by Dr. Lempert, case A. K.

a result of otosclerosis, and that it is still unaltered after the fenestration operation. Recently, however, a few writers have reported small losses of bone-conduction acuity in otosclerosis and corresponding gains after fenestration. Thus Woods[17] found his bone-conduction tests to show a progressive loss of high-tone acuity as characteristic of the early stage of otosclerosis, and this loss was recovered, at least in part, after fenestration. He reported an average recovery in the middle frequencies of 11.4 db.

In relation to these results we may recall that in Smith's experiments on the cat[8] a mechanical fixation caused a moderate reduction in the responses to bone-conducted sounds. The effect was especially marked in the region of frequency around 700 cycles, where it reached an average value of 20 db. This region is evidently the region of mechanical resonance of the ear.

We recognize two principal means of cochlear stimulation by bone-conducted sound. One is the compressional method, in which the sound produces alternate expansion and contraction of the cochlear capsule, and the other is the translatory method, in which the sound moves the cochlear capsule bodily back and forth.[18]

Let us consider the situation first in the normal ear. In the compressional method of stimulation, as illustrated in Fig. 9, a contraction of the capsule forces the incompressible cochlear fluid out-

ward by way of the oval and round windows, and an expansion draws it back. However, these fluid movements are asymmetrical with respect to the basilar membrane. The fluid cavity on the vestibular side of the membrane, comprising as it does the scala vestibuli, cochlear duct, and adjoining labyrinthine spaces, exceeds by a good deal—by a ratio of 5 to 3—the cavity on the tympanic side, which is only the scala tympani. Also, because the oval window is attached to the ossicular mechanism it presents a certain impedance to the displacement. Accordingly, a large fraction of the displacement takes place across the basilar membrane in the direction of the round window, and this membrane is forced to move back and forth, whereby its cells are stimulated.

In the translatory method of stimulation by bone-conducted sound, as Fig. 10 illustrates, the cochlear capsule is effectively shaken back and forth. The inertia of the fluid contents causes them to tend to lag behind. When the motion is in line with the oval and round windows, or has a component in this direction, the fluid can move alternately toward one window and the other, now bulging out the membrane of the round window and then the footplate of the oval window. The basilar membrane lies in this path of fluid movement and is subjected to displacement in the process.

In both these means of stimulation the middle ear mechanism plays an incidental role. In the compressional method the impedance presented to the fluid movement by the oval window varies with frequency according to the mechanical resonance characteristics of this mechanism. In the translatory method likewise, and for the same reason, this mechanism imposes its characteristics upon the fluid movement.

When in otosclerosis the oval window becomes occluded the compressional method of stimulation by bone-conducted sound is still effective. Indeed, it ought to be even more effective than normally, for now none of the displaced fluid can find relief by way of the oval window and all must be squeezed toward the round window. The basilar membrane should be vigorously stimulated. On the other hand, the translatory method of stimulation now largely fails. It fails for the same reason that aerial sound applied to the round window is relatively ineffective in displacing the cochlear fluid. The fluid simply moves with the capsule and so does the basilar membrane. The alteration of form of this membrane, which is essential

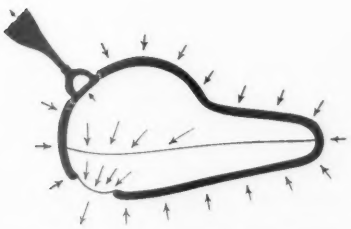

Fig. 9.—Bone-conduction stimulation in the normal ear by the compressional method. The cochlea is pictured as composed of two chambers separated by the basilar membrane. The larger chamber contains the oval window, in which the stapes rests, and the smaller chamber contains the round window. A compressional stimulus consists of a pressure exerted upon all parts of the cochlear capsule, and is represented by the outside arrows. Such a stimulus reduces the volume of the capsule and displaces fluid outward in the direction of the two windows, but more toward the round window because it presents the smaller resistance. Displacement at the oval window is partially restrained by the stapes and its connection with the ossicular mechanism. That part of the fluid in the larger chamber that is moved toward the round window causes a displacement of the basilar membrane downward. When an instant later the vibration changes its direction and the compressional phase is replaced by an expansion phase all these actions are reversed and the basilar membrane is displaced upward.

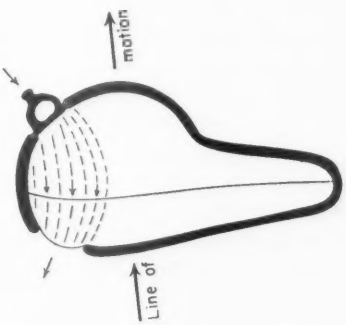

Fig. 10.—Bone-conduction stimulation in the normal ear by the translatory method. The effective stimulus is a bodily motion of the cochlear capsule in line with the two windows, which is roughly along the vertical axis of the drawing. If at a given instant this motion is upward, as the large arrows suggest, the fluid will lag behind, and accordingly the oval window will bulge inward, the round window will bulge outward, and the basilar membrane will be displaced downward A little later, when the stimulus motion is in the contrary direction, the fluid movements will likewise be reversed and the basilar membrane will be displaced upward.

to stimulation, is now minimized. Because one of these means of stimulation is enhanced and the other is reduced we should expect an altered picture of bone-conduction sensitivity, but it is difficult to predict what new form it will take. It is reasonable to expect a variation with frequency and with the particular techniques used in applying the stimulus.

When the otosclerotic ear is fenestrated the bone-conduction situation returns in the direction of the normal, but without the reactive complications presented at the oval window by the middle ear system.

THE COURSE OF OTOSCLEROSIS

In this discussion we have identified three primary changes in the operation of the conductive mechanism as a result of the otosclerotic lesion, namely, the mechanical fixation of the ossicular chain, the loss of the transformer system, and the immobilization of the cochlear fluid. To these under certain conditions are added further complications in the form of phase interactions between alternative pathways of entrance to the cochlea. We have now to consider how these physical conditions vary in the course of the disease and modify the ear's efficiency in sound conduction.

The progressive nature of otosclerosis has already been referred to. As is well known, it has a gradual, even an insidious, onset, and for a long period the depreciation of hearing will hardly be recognized. Eventually, a clear awareness comes to the person that a loss of hearing has occurred. Medical advice is not usually sought until this loss amounts to 40 db or more, when the person begins to be definitely handicapped in his social relations. From a physical standpoint this is a serious impairment, for a 40 db loss represents a reduction in the conduction of acoustic energy to one ten-thousandth of its normal value.

From histological studies (e.g., Guild[19]) we can infer the early course of events. The first conductive impairment probably appears when a bridge of bone forms at some point on the annular ligament of the stapes and produces a firm connection between the stapes footplate and the cochlear capsule. The impairment progresses as this bridge is strengthened and widened and as others appear at other places on the ligament. The end stage is that in which the stapes footplate is completely ankylosed and the oval window is obliterated.

We can now attempt a theoretical analysis of the physical conditions governing the ear's action at different stages of the disease.

In the early stage, when the loss of transmission is no more that 20 db., the physical effects are relatively simple. We have only to deal with the mechanical fixation of the ossicular mechanism. The sound is still transmitted by way of this mechanism and the transformer action is maintained. The loss results because the increased stiffness that is reflected to the drum membrane causes less sound energy to be taken up by this membrane, and because much of the energy that is taken up by it is wasted in overcoming the increased frictional resistance and in producing vibrations of the bony capsule.

As the fixation approaches 30 db the picture grows more complicated. The fixation effect now cancels the advantage to conduction afforded by the transformer action, and the ossicular route to the cochlea is no better than the aerotympanic route through the round window. With the two routes operating with much the same efficiency we have to consider phase interactions between them. In general, because the middle ear mechanism retains its phase changing characteristics, the interactions will have only slight effects over most of the auditory scale, but at certain frequencies we can expect destructive interference. Also, because the phase action of the middle ear is somewhat unstable, subject to modification by simple changes in the structures, we can expect the audiograms to show rather wide variations from time to time.

As the fixation exceeds 30 db the round window route to the cochlea becomes the one of importance. The loss of hearing should now proceed relatively slowly for a time because the only progressive change is the increasing stiffness conveyed to the drum membrane and its barrier effect toward the entrance of sound waves to the tympanic cavity.

Then, as the oval window finally is closed by the lesion, we have the ultimate and most grave effect, which is the immobilization of the cochlear fluid. This immobilization negates the effectiveness of the round window route and rapidly drops the hearing to new depths.

The fenestration operation, as we have seen, effects a re-mobilization of the cochlear fluid. Also, when carried out by the Lempert method, it removes the barrier presented by the stiffened drum mem-

brane and gives the sound waves a readier access to the cochlear fluid. The transformer action formerly provided by the middle ear mechanism of course is not regained.

Because the transformer action is lost we must expect the fenestration operation to restore the hearing only to a point within about 28 db of the normal. This is indeed the usual result, as shown for example in a recent study by Davis and Walsh,[20] in which they found an average acuity of 27 db below the zero mark of the audiometer in the fenestrated ears.

Sometimes after fenestration the hearing is decidedly better than this: it returns to 20 db or 10 db or even closer to the audiometric zero. This fortunate result does not represent an error of measurement, but is real: it is repeatedly shown by the hearing tests. Therefore it has to be explained in view of the loss of the ossicular mechanism which as we know depreciates the efficiency of the ear by 28 db.

It appears from the results presented earlier in this paper on blocking the path to the round window that a part of this unexpected gain can arise from a favorable phase interaction between fenestral and round window routes of sound. However, this condition can account at most for a gain of only 6 db.

I think the main explanation of this unexpected gain is simply that these persons originally had better than average hearing and have now been returned to a level about 28 db below their former one.

PRINCETON UNIVERSITY.

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XCI

LESIONS OF THE EXTERNAL AUDITORY CANAL

WALTER P. WORK, M.D.

SAN FRANCISCO, CALIF.

Nowhere in the English otorhinolaryngological literature has it been possible to determine in a systematic manner what diseases should be included in a discussion on lesions of the external auditory canal. It is true that the textbooks describe and classify eczema, fungus infection, furunculosis, nonspecific external otitis and similar conditions in detail but little else in sequence is noted. This clinical study was undertaken to bring together isolated information, to present illustrative cases showing lesions of the external auditory canal and to classify these lesions in a logical manner according to:

I. Those arising directly in the external auditory canal.

II. Lesions or mechanical forces that involve the external auditory canal secondarily by extension, encroachment or contact.

III. Those due to developmental disturbances.

Only twice since the founding of the American Otological Society in 1868 have lesions of the external auditory canal been discussed briefly before this group. Bryant[1] at the meeting of the Society in Washington, D. C., in 1903, reported that he had removed a small lesion of the external auditory canal which proved to be fibroid in character. One year later at the Society's meeting in Atlantic City, New Jersey, Pooley[2] reported a case of fibroma of the mastoid, auricle and auditory canal. Clinically, it appeared to be a sarcoma. Microscopically, it was also reported as fibroid tissue. (However, it is noted that Robertson[3] at the meeting of the American Otological Society at Newport, R. I., in 1870, presented a case that may have been a sarcoma of the external auditory canal in a female patient. From his report the exact location and nature of the lesion is not clear.)

From the Department of Surgery (Otolaryngology Section), Stanford University School of Medicine, San Francisco, California.

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During the past few years more interest has developed in the external auditory canal because of better diagnostic methods including such techniques as the tomograph, widespread use of biopsy, tissue cell diagnosis as in Papanicolaou's method and section of the temporal bone including the external auditory canal for microscopical study. Not only have diagnostic methods improved but surgical techniques as well have shown remarkable advancement. Thus, bony exostoses, traumatic or even congenital atresias can be corrected surgically with more hope of success than at any other previous period of time. The use of sulfa drugs and antibiotics must also share in the brilliant surgical successes for no doubt in many former cases where poor results were obtained, infection was to blame rather than the surgical procedure.

I. Lesions Arising in the External Auditory Canal.

A. NEOPLASMS OF EPITHELIAL ORIGIN.

1. BENIGN TYPES.

a. *Nevi.* The integument of the external auditory canal contains sebaceous glands, ceruminous glands, sweat glands and hair follicles. These structures are characteristically found in the outer portion of the external auditory canal. The skin over the bony canal becomes thin, more delicate, free of glandular structure and hair follicles. Although not common, benign epithelial lesions can arise in the external auditory canal. They vary from the small, smooth, epithelial nevus[4] to palliomata with a horny, warty surface[5] and are usually asymptomatic unless externally large when symptoms of blockage may arise. They have a pedunculated or a sessile base and may or may not contain pigment. Treatment, if indicated, is by electrocautery removal using care not to destroy so much normal tissue that stricture will result.

2. MALIGNANT TYPES.

a. *Squamous Cell Carcinoma.* The most common type of malignancy of the external auditory canal is the squamous cell carcinoma and its variations the prickle cell or the basal cell carcinoma. Even though this is the most frequently occurring lesion of the external auditory canal, it in itself is a rare condition. Fraser[6] reported that of 646 cases of diseases of the external meatus observed between 1906 and 1929 only 3 cases were of malignant disease. In

the same report Fraser[6] points out that at the Royal Infirmary out of 6,605 cases in which there were affections of the external auditory meatus, only 13 cases showed malignant disease. Robinson[7] reported that over a 20-year period from 1905 to 1924 at the Manhattan Eye, Ear, Nose and Throat Hospital, only 48 cases were diagnosed as tumors of the ear although 212,000 ear cases were admitted. Of these 48 cases the external ear and the auditory canal were involved primarily in 19. The highest incidence of cancer arising in the external auditory canal was reported by Broders[8] in 1921 wherein 14.28% of 63 cases of cancer of the ear showed some type of epithelial malignancy of the external auditory canal. At the San Francisco City and County Hospital during a recent 16-year period, there were 16,375 eye, ear, nose and throat cases admitted and significantly there were no cases of primary squamous cell carcinoma of the external auditory canal among them. However, there were 26 cases of squamous cell carcinoma involving the pinna. Squamous cell carcinoma of the external auditory canal is reported by Fineberg and Jorstad,[9] Land,[10] Gibb,[11] Risch and Lisa,[12] Barnes,[13] Dundas-Grant,[14] Politzer[15] and others.[16-23] The largest series presented was by Fraser[6] who reported that he had collected 12 cases.

Squamous cell carcinoma arising in the external auditory canal appears to affect males as frequently as it does females. The disease is characterized by its slowness of evolution and the lack of early symptoms. The tympanic membrane may be intact early in the disease but it can be involved by direct extension, blockage and infection. Primary squamous cell carcinoma of the canal may also arise in conjunction with a suppurative otitis media.

The patient may seek help only because of mild otorrhea or slight deafness. Upon examination of the ear there is a mass located in the external auditory canal that bleeds easily with manipulation. If the cancer has progressed, pain is characteristic and is extremely severe. Invasion of the middle ear may be associated with more profound deafness and copious foul otorrhea. Further local extension from the primary site in the external auditory canal may denote involvement of bone or vital structures. Invasion of the facial nerve, labyrinth, cochlea, mastoid process, temporomandibular joint, sigmoid sinus, jugular bulb, dura and brain may be considered the result of direct local spread. Involvement of any or all of these structures inherently carries a poor prognosis. Metastasis to regional lymph glands

is slow to take place, and the first group of nodes usually involved are the pretragal group, the superficial and deep cervical nodes being involved later. Widespread distant metastases are rarely seen since if the primary lesion is unchecked death occurs from local extension to vital structures before such metastases occur.

The diagnosis of squamous cell carcinoma of the external auditory canal must be early if the prognosis for cure is to be good. Suspicious appearing lesions of the external auditory canal should be considered cancer until proved otherwise. Biopsy with microscopical examination of the tissue removed should be repeated as often as necessary to establish a correct diagnosis. The Papanicolaou method of cell staining and microscopical examination if used judiciously may lead to even earlier diagnosis.

If the lesion is extensive, treatment with surgical excision alone offers a poor prognosis. If at the time of surgical excision it is determined that the neoplasm has not been removed, the procedure of choice is to employ supplementary irradiation immediately. On the other hand, if at the time of surgical excision it can be determined by direct inspection and frozen section technique that the lesion has been removed adequately, it is the procedure of choice not to employ supplementary irradiation until recurrence has been proved. It is inferred from this statement that regardless of whether the approach is postauricular or endaural, a wide margin of normal tissue must be included in the resection. If lymph gland metastases have occurred, they should be removed radically (including neck gland dissection if necessary) at the primary operation.

The following case illustrates some pertinent points:

CASE 1.—F. E., female, aged 86, was operated endaurally January 17, 1947, for squamous cell carcinoma of the right external auditory canal. Her history was notable in that she had had chronic right otorrhea and deafness for six years. Severe pain in the ear had been present several months. She had consulted numerous physicians without help. A granular mass was noted in the right external auditory canal attached anteriorly and inferiorly. It bled with manipulation. There was a mixed hearing loss in the right ear. Labyrinthian studies and neurological examination were normal. At operation the neoplasm was found to invade the nonarticular bony surface of the temporomandibular joint. The tympanic membrane was perforated and the epitympanum was filled with cancerous appearing tissue. A thorough radical mastoidectomy was done including removal of the nonarticular surface of the temporomandibular joint down to the eustachian tube orifice. The postoperative course was uneventful. There was good healing of the wounds. However, seven months later there was a recurrence of the lesion in the canal as shown by biopsy and she was immediately given 4,990 roent-

gens of x-ray therapy as measured in air to the right ear. Her health has been excellent and there was no evidence of recurrence as determined on February 20, 1950, by gross inspection and by Papanicolaou's technique. She hears a loud shout in the operated ear. The mastoid cavity is clean and partially filled with scar tissue with a small opening at the meatus leading into the remnants of the middle ear space. Labyrinthian tests by the Dundas-Grant technique showed normal response bilaterally.

Comment. The patient had symptoms for six years before the diagnosis was made. One of the chief symptoms was severe pain in the ear. At surgery local extension of the lesion was found anteriorly involving the nonarticular surface of the temporomandibular joint. Surgery and roentgen therapy were employed in this case to effect a 37-month arrest of the neoplasm.

b. *Adenocarcinoma.* Adenocarcinoma of the external auditory canal is extremely rare, in fact, so rare that only an occasional case report is noted in the literature. In 1936 Lukens[24] reported a case of adenocarcinoma of the external auditory canal. A female, aged 55, was treated by removing the lesion with a snare and follow-up radio-ionization. Fraser[6] reports two cases of malignant sebaceous adenoma of the external auditory canal. Blair et al[25] also report a case of malignant sebaceous adenocarcinoma of the external auditory canal. It is important for these lesions to be recognized and biopsied early. The treatment of choice is surgical excision since they are resistant to roentgen irradiation.

B. NEOPLASMS OF SUPPORTING TISSUE ORIGIN.

1. BENIGN TYPES.

a. *Hyperostoses.* The most frequently encountered benign types of lesions arising in supporting tissue of the external auditory canal are the bony hyperostoses. Burton[26] points out that exostoses of the auditory canal were found in prehistoric skulls and presents photographs showing some of the larger types of lesions. In fact, as Burton notes, in some collections of skulls a rather high incidence has been discovered.

Hyperostoses are not frequently found but if the large or small ones noted on a routine examination were recorded, their incidence would probably be found to be greater than anticipated. One is familiar with the small exostoses which occur on the innermost portion of the bony canal that are asymptomatic and require no treatment.

In general there are two types of hyperostoses; namely, those with a small and those with a sessile base. For clarity and uniform-

ity those of the former group will be considered osteomata while those in the latter group will be considered exostoses.

The etiology of these lesions is not known but it is postulated that they arise from chronic irritation as in suppuration of the middle ear and eczema of the canal. Disturbances of embryological development, irritation from trauma and hereditary factors have also been suggested. Males seem to be affected more frequently than females.

The symptoms vary from slight to partial deafness, fullness in the ear, otorrhea, pressure from retained cerumen and cell detritus and pain. Often the lesions are discovered on routine examination.

Indications that demand surgical removal are: large size, otorrhea from local irritation, otitis media or mastoiditis, hearing loss, pressure symptoms, wax and cell detritus accumulations, and recurrent episodes of pain.

(1) Osteomata. An osteoma may vary from that of a small millet seed to over 2 cm in diameter. In 1948 Belal[27] reported a case of an osteoma protruding from the right external auditory canal which when removed measured 2 cm by 1.5 cm.

Osteomata of the external auditory canal may be removed through the canal if they are not too large. The pedicle of the osteoma is fractured with a curette and the osteoma is lifted out. Care must be taken not to injure the eardrum. If there is a remaining spine of the pedicle, it may be smoothed with a small polishing burr.

The following two cases represent lesions of this type:

CASE 2.—J. A., male, was admitted to the hospital on July 21, 1947, complaining of a stopped-up feeling of the right ear following a recent cold. Examination of the right external auditory canal showed it to be filled with a bony mass attached to a pedicle superiorly and posteriorly. Under general anesthesia on July 24, 1947, the osteoma was removed with a curette (Fig. 1). The remaining 2-mm pedicle was smoothed with a small polishing burr. Convalescence was uneventful.

CASE 3.—E. L., male, complained of fullness in the right ear for 12 to 18 months which was always worse following swimming. Examination showed that the right ear canal was entirely occluded by a mass attached to the posterior superior canal wall. On January 2, 1947, an osteoma was removed from the right external auditory canal by fracturing the base of it with a curette. A polishing burr was used to smooth the pedicle surface. Convalescence was uneventful.

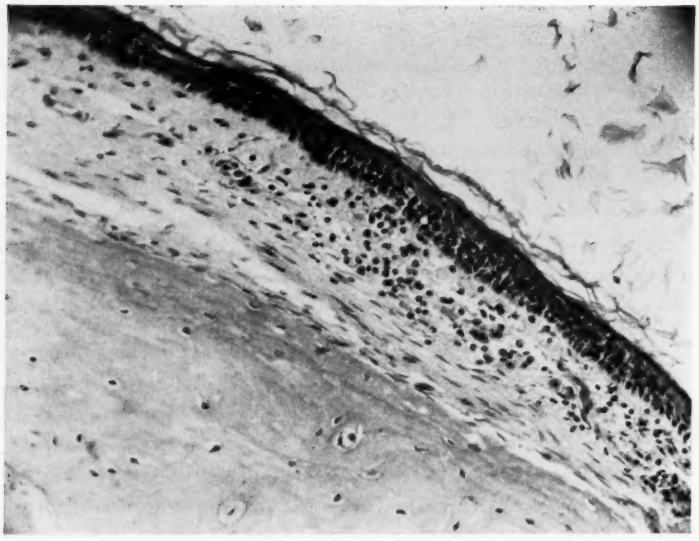

Fig. 1, Case 2.—Photomicrograph (x 200) showing a portion of the osteoma removed from the external auditory canal. Note the squamous epithelium and subcutaneous tissue overlying dense bone. (x 200)

(2) Exostoses. The surgical removal of exostoses presents a different problem than the removal of osteomata because of the likelihood of stricture forming later at the operative site. The exostoses may be surgically approached by a posterior auricular incision. However, following the instruction and technique of Lempert it has been shown that they may be removed successfully through a single endaural incision. The skin of the canal is dissected free from the exostosis and the bony growth is removed with a small polishing burr throughout its length in the external auditory canal. A portion of the mastoid cortex and mastoid cells may be removed if indicated. An anterior modification of the incision must be carried out if the attachment of the exostosis is found in the region of the nonarticular surface of the temporomandibular joint. Once the lesion is exposed in this area removal proceeds as before.

Case 4.—M. E. G., female, had an exostosis of the inferior posterior surface of the left external auditory canal. There was discomfort, pain and a mild loss

of hearing in the ear for a period of one year. On July 6, 1949, removal of the exostosis of the external auditory canal was done through a single endaural incision. The exostosis was removed subepithelially to within 2 mm of the bony annulus tympanicus. A small portion of mastoid cortex was removed also but the mastoid cells were not entered. Healing was per primum and a satisfactory result was obtained. Hearing returned to normal.

b. *Fibroma.* Fibromata of the external auditory canal arise in supporting tissue. Bryant[1] reported a case of fibroma of the external auditory canal as shown previously. Simple careful removal apparently cured the patient. Lowy[4] reported the case of a male who had a fibroma in the right external auditory canal. Local removal was followed by a recurrence of the lesion which the author proposed to treat further with irradiation. The same author also records a case of pseudomyxomatous fibroma treated with radium.

c. *Xanthoma.* An interesting and certainly a most rare case was reported by Rosenberger[28] in 1937 of a xanthoma of the external auditory canal. He removed the lesion completely by biopsy. It was reported by the pathologist to be a xanthomatous type of lesion, probably of inflammatory origin. The patient was well following its removal.

d. *Endothelioma.* In 1922 Layera[29] reported that he removed a lesion from the left external auditory canal of a patient. The base bled profusely after it was evulsed. Microscopical examination showed it to be an angio-endothelioma. There was no follow-up pertaining to this patient.

e. *Lipoma.* Bull[30] reportedly removed a lipoma from the external auditory canal of a patient.

f. *Chondroma.* Konietzko[31] reported a case of a 4-mm club-shaped lesion attached by a stalk to the anterior bony auditory canal wall which upon removal proved to be a chondroma by microscopical examination. It is of interest to note that this lesion did not arise in the cartilaginous meatus but in the bony canal.

2. MALIGNANT TYPES.

a. *Sarcoma.* A giant cell sarcoma of the external auditory canal complicating mastoiditis was reported by Herbert[32] in 1923. A male patient, aged 53, had a growth removed from the auditory canal at the time a mastoidectomy was done. It proved to be malignant as designated above. It was treated with radium but there was no indication in the report as to the permanency of cure.

b. *Angiosarcoma.* Politzer quotes Milligan[33] as presenting a case of angiosarcoma with caries of the walls of the meatus.

c. *Osteosarcoma.* Scheibe[34] reported the case of a 17-year-old male with a bean sized tumor attached to the anterior bony canal wall by a stalk. Microscopical examination after removal showed it to be an osteosarcoma.

d. *Fibrosarcoma.* Buck[35] reported the occurrence of a fibrosarcoma in the external auditory canal of a patient.

It is interesting to note the number of different types of tumors reported arising from supporting tissue as contrasted with those arising from epithelial tissue. Other lesions that develop within the external auditory canal such as furunculosis, eczema, blebs, diffuse bacterial infections, erysipelas and fungus infections are beyond the scope of this discussion but are mentioned here only to complete this category of classification.

II. Lesions or Mechanical Forces That Involve the External Auditory Canal Secondarily by Extension, Encroachment or Contact.

A. X-RAY BURNS.

1. ROENTGEN THERAPY.

It is well known that it is uncommon for x-ray therapy to produce injurious changes in bone through intact soft tissues as they tend to filter a certain amount of the damaging rays. Apparently, one notable exception to this is the external auditory canal where bone is found to lie in close proximity to the overlying skin. The damaging x-ray therapy is usually directed at other lesions in close proximity to the external auditory canal, such as: carcinoma of the pinna, carcinoma of the nasopharynx, carcinoma of the face or metastatic malignant glands. To treat such neoplasms satisfactorily by roentgen therapy the external auditory canal of necessity may fall in one of the portals used. In the cases reported in this study the dosage of x-ray therapy to the primary lesions was in excess of 4000 roentgens as measured in air. During the course of irradiation therapy there was no difficulty in the external auditory canal but several months following the completion of the therapy, the patients complained of pain in the ear and foul otorrhea. Inspection showed that the canal was filled with a purulent exudate and when this was re-

moved, bare bone could be seen and palpated in the bony canal wall inferiorly. The bone would not bleed with manipulation and if watched over a period of weeks or months there were no granulations formed on its surface. Epithelium about the bone appeared to be pearly and piled up. The epithelial borders bled readily with trauma, and pain was extreme on touch or pressure. The tympanic membrane was not noticeably involved by the therapy.

During the course of irradiation therapy, changes in the vascular supply and changes in the marrow spaces of the bone may occur. If the therapy is prolonged, aseptic necrosis of the bone will result. This in turn causes a denudation of the thin skin of the bony external auditory canal wall leading to chronic ulceration. If the portal of entry of the roentgen therapy is large and the dosage is sufficiently concentrated, the squama of the temporal bone and the head of the mandible may be affected by aseptic necrosis.

Ulceration and bony necrosis can be alleviated only by wide surgical intervention. Sedation of the patient and local cleanliness may be all that is indicated in far advanced cases when the neoplasm is known to be fatal.

CASE 5.—J. W. P., male, aged 63, completed a course of roentgen therapy to a small basal cell carcinoma anterior to the right tragus and adjacent part of the face in November, 1936. He received a total of 6000 roentgens as measured in air. Several months after completion of the course of therapy, the patient developed a right otorrhea with a foul odor and pain. He was treated conservatively over a period of several years and in February, 1942, entered the San Francisco City and County Hospital. Because of the extreme foulness of the discharge and the finding of bare bone in the external auditory canal a radical mastoidectomy was performed in March, 1942. Included in this operation was removal of the condyle of the mandible and a portion of the root of the zygomatic arch. All removed bone showed aseptic necrosis. The area was covered with a split thickness skin graft which did not live. Since the defect was so large a full thickness pedicle graft was created on the right side of the patient's neck. In July, 1942, further excision of bone in the areas noted was done. By successive stages the pedicle graft on the neck was shifted upward to the anterior part of the defect.

The patient's condition remained satisfactory for the next seven years. There was no evidence of recurrence of the carcinoma although there remained some suppuration within the operative cavity. There was no apparent disability from removing the condyle of the mandible. During this period of time a facial paralysis developed probably due to local edema about the seventh nerve, but owing to the patient's age he was not subjected to further surgery. Death occurred from generalized arteriosclerosis and senility on January 5, 1949. At autopsy there was considerable softening in the squamous portion of the temporal bone and there was a dural exposure of the old operated site. There was no evidence of local recurrence of the neoplasm.

Comment. This is an example of severe necrosis of the bony external auditory canal and related structures wherein surgical intervention was instrumental in giving the patient a few years of semicomfortable living.

CASE 6.—W. Y., male, Chinese, aged 53, was seen in Stanford University Hospital Clinic in 1944 because of blindness O.S. and a diagnosis of retrobulbar tumor was made. A left frontal parietal exploration was done and a tumor was found about the optic nerve. This proved to be a squamous cell carcinoma probably arising in the nasopharynx. (The nasopharynx had been previously searched for neoplasm without success.) Between August and September, 1944, he received 4000 roentgens of therapy to the left side of the head. On June 6, 1945, an enucleation of the O.S. was done. In July, 1947, he received 2790 roentgens as measured in air to each side of the neck for cervical metastatic glands. On November 6, 1947, exposed bone of the left external auditory canal was noted. At this time there was a foul otorrhea and pain in the ear. He was treated conservatively. In August, 1948, a biopsy of the skin about the ulcerated area of the left external auditory canal was reported as showing no malignancy. In April, 1949, the patient was seen for the first time in private practice. Inspection showed bare bone to be present inferiorly in the external auditory canal extending throughout the bony portion of the canal. The nasopharynx was re-biopsied and squamous cell carcinoma of the nasopharynx was found. He was given further irradiation therapy. With conservative treatment the foul otorrhea in the left external auditory canal improved although there was no basic improvement in the ulceration.

Comment. This case illustrates the type of lesion that can be produced in the external auditory canal by roentgen irradiation to the nasopharynx. Because of the poor over-all prognosis in this case, no operative intervention was indicated.

2. RADIUM EMANATION.

The end result of radium emanation in bone is much the same as with x-ray therapy. In 1926 Phemister[36] showed that by implantation of radium into the bones of dogs, areas of aseptic necrosis could be produced. Radium placed in the external auditory canal can cause aseptic bony necrosis and clinically the lesion appears to be the same as that produced by roentgen therapy. Although the following patient's record is not available, it is well remembered that while in the Army Service, a Colonel was examined. He was complaining of pain and otorrhea of his left ear. Upon inspection of the ear there was bare bone in the external auditory canal and ulceration inferiorly. The ulceration was due to aseptic necrosis of a portion of the tympanic bone. There was an otitis media with a perforation of the tympanic membrane. Biopsy of the epithelium about the ulceration showed no evidence of neoplasm. He stated that 19 years previously radium had been placed for many hours in the external auditory canal for polyps of the middle ear. His disability was so great from this lesion that he was unfit to continue in military service. This case illustrates the serious nature of the re-

sulting disability from overzealous treatment of a relatively innocuous lesion.

Aseptic necrosis with overlying ulceration of a portion of the bony external auditory canal wall is similarly produced by roentgen therapy or radium and the lesions cannot be differentiated clinically. These lesions produce severe disability from pain, otorrhea, worry and loss of sleep.

B. ATRESIA OF THE EXTERNAL AUDITORY CANAL DUE TO OTITIS MEDIA.

Strictures or stenoses of the external auditory canal may occur coincident with suppuration of the middle ear and mastoid, infection of the cartilage, and external otitis. They can eventually lead to a narrowing of the external auditory canal in localized areas or throughout its length. Operative procedures and trauma can be followed by stricture. These lesions are due to a thickening of the skin or to a thickening of the underlying subcutaneous fibrous tissue or both. Most strictures cannot be successfully corrected unless the underlying pathology is removed.

It is rare indeed for the stricture to be complete and even more rare for the remaining patent canal to be bridged by a membrane.

The following case illustrates the latter condition:

CASE 7.—E. F., male, aged 24, complained of bilateral otorrhea for as long as he could remember. He stated that his ears drained almost continuously until 1943, at which time there was cessation of discharge. In July, 1946, while in the Army he was told that he had a closure of both external auditory canals. He stated that the membranes of both canals were lanced and that he heard better for a period of six to eight months. The ears remained free of discharge. The patient was admitted to the hospital on January 17, 1950. He gave the above history and stated that he desired to have something done about his ears so he could hear better. There was a conduction hearing loss of 30 db in the right ear and 25 db in the left ear. Bone conduction, bilateral, was normal. Examination showed a thin membrane occluding the right and left external auditory canals at the junction of the bony and cartilaginous portions. These membranes would move outward when the patient was asked to hold his nose, close his mouth and blow. X-ray films of the mastoids showed a diploic type with some cellular structure present. Labyrinthian turning tests were normal. On February 21, 1950, under general anesthesia an endaural exposure of the area on the right was done. A partial mastoidectomy and removal of the posterior bony canal wall were completed thus exposing the atresia (Fig. 2, *above*). The membranous atresia was severed and a flap was created as one does in the fenestration technique (Fig. 2, *below*). A large central perforation of the drum was present. The middle ear was clean. The incus, malleus and stapes were present and appeared to be intact.

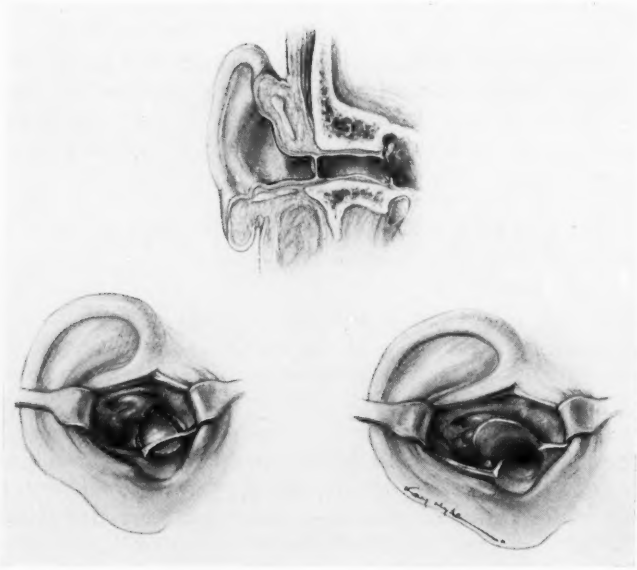

Fig. 2, Case 7.—*above*: Schematic representation of the occluding membrane of the right external auditory canal as seen in coronal section. Note large perforation of tympanic membrane explaining movements of the false membrane during Valsalva's maneuver.

Below, left: Drawing showing dotted line along which the membranous atresia and the meatal flap were cut.

Below, right: Drawing showing meatal flap after the membranous atresia was excised and just before completion of the incisions so that the flap would lie in correct position.

No more was done to the middle ear than to scarify the borders of the remaining portion of the tympanic membrane. The flap was packed posteriorly as in fenestration technique. Postoperatively there was an average 9 db improvement in hearing throughout the speech frequencies.

Comment. This case illustrates one of the rare secondary lesions of the external auditory canal following suppuration of the middle ear. Its correction surgically was feasible since the middle ears had been free of suppuration for seven years. The Valsalva maneuver showed that the tympanic membranes were perforated. The surgical technique employed was not unlike the technique used during the war years to correct certain suitable cases of traumatic atresia. Operation is planned for the other ear.

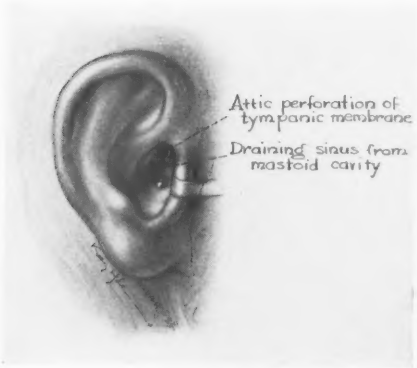

Fig. 3, Case 8.—Drawing showing right ear and auditory canal. Note the draining sinus opening into the posterior part of the bony canal. A probe could be passed deep into the mastoid proper. Inspection through the opening showed gross pulsations of the discharge denoting areas of dural exposure.

To postulate how these false membranes developed might be of interest. Due to long continued suppuration of the middle ear, the external auditory canals were bathed in pus. Local irritative reactions were set up at the junction of the cartilaginous and bony portion leading to early stricture. By creating an ulceration about the circumference of the auditory canals, granulations formed that eventually led to false membranes. A large part of the occlusion was fibrous tissue, the false membrane being relatively small. This patient probably had a stricture for years before the final atresia developed. Most unusual, too, is that a simultaneous process involved both ears.

C. MASTOID PATHOLOGY CAUSING LESIONS IN THE EXTERNAL AUDITORY CANAL.

During the years when mastoiditis was prevalent and particularly when acute mastoiditis occurred, it was not unusual to see secondary inflammatory lesions of the bony external auditory canal wall. One form of lesion was a periostitis with drooping of the canal wall. Another variation in these secondary lesions was the formation of a mass in the bony canal which appeared clinically to be a furuncle. With careful inspection, manipulation and the palpation of bare bone deep within the area, the correct diagnosis could usually be determined. Sometimes a probe could be passed directly into the mastoid cavity. Careful simple mastoidectomy often effected relief.

Fig. 4, Case 8.—Note the completeness with which the cholesteatoma had eroded and destroyed the right mastoid. This film was taken before operation.

A more uncommon lesion is the erosion of a cholesteatoma into the bony external auditory canal from the mastoid cavity forming an epithelialized tract.

The following case illustrates such a series of events:

Case 8.—A. G., male, 61, was first seen in 1947 in private practice complaining of discharge from the right ear. He stated that his ear had been draining for a period of 30 years or longer. During this period of time the ear had been dry on many occasions without evidence of difficulty except hearing loss. Two years before this consultation the ear started to discharge more profusely. The patient's examination showed that the drum had a posterior superior marginal attic perforation with some discharge in it and a small amount of granulation tissue about its periphery. A 1-cm opening through the bony external auditory canal leading directly into the mastoid cavity was found (Fig. 3). X-ray films showed a very large cholesteatoma completely destroying the mastoid (Fig. 4). Neurological clearance was obtained. Bilateral hearing loss was of a mixed type. Ophthalmological examination was normal. On November 20, 1947, a radical mastoidectomy was done through a posterior auricular incision. A large cholesteatoma was found protruding through the epidermized sinus tract into the external auditory canal

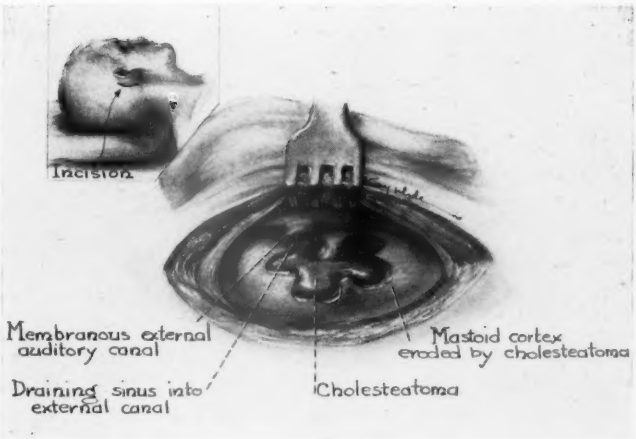

Fig. 5, Case 8.—Extensive erosion by the cholesteatoma of the bony external auditory canal is depicted as well as massive erosion of the mastoid cortex.

and the mastoid cortex was partially eroded (Fig. 5). The cholesteatoma was removed and the radical mastoidectomy completed, care being taken not to injure the exposed facial nerve (Fig. 6). A Koerner meatal plastic flap was created and the posterior auricular incision was closed tightly. The patient's postoperative course was uneventful. Two and a half years later the mastoid cavity appeared healed, dry, and well epithelialized.

Comment. The cholesteatoma apparently had been forming for many years. It was of the secondary type having grown through the epitympanum and aditus destroying the whole mastoid process and eventually eroding and forming an epithelialized fistulous tract through the posterior bony external auditory canal wall. Surgical correction has proven satisfactory.

D. NEOPLASMS ORIGINATING ELSEWHERE AFFECTING THE EXTERNAL AUDITORY CANAL.

The most common neoplasm to encroach upon the external auditory canal by direct extension is the squamous cell carcinoma of the pinna. As noted previously, 26 cases of squamous cell carcinoma of the pinna were admitted to the San Francisco City and County Hospital during a recent 16-year period. In only one of

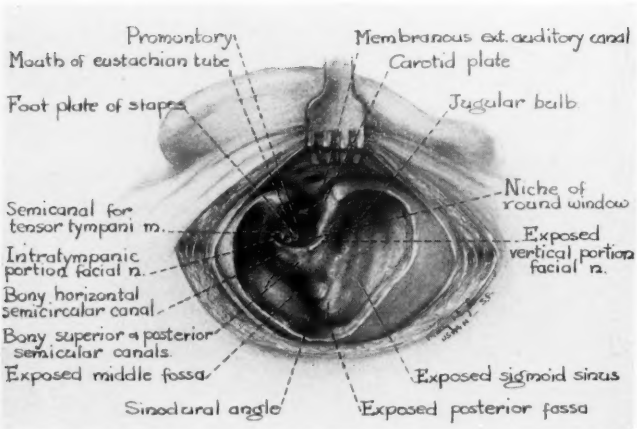

Fig. 6, Case 8.—The cholesteatoma had done most of the mastoidectomy in this patient. The sinus tract and the bony canal wall were removed surgically.

these cases was there an indication that the external auditory canal was involved by neoplasm through direct extension. Wide surgical excision of the primary lesion was made more difficult by this extension.

Other malignant lesions such as primary sarcoma of the pinna may extend into the external auditory canal. Neoplasms of the parotid gland may involve the canal by direct extension particularly if showing malignant degeneration.

Malignant neoplasms arising in the nasopharynx may encroach upon the external auditory canal by direct extension along the eustachian tube to the middle ear and thence through the tympanic membrane to the canal wall.

Extension of meningiomas through the bony squama of the temporal bone with encroachment upon the superior portion of the external auditory canal in a patient was reported by Woltman and Love.[37] Nurenberg, Peters and Wagenhauser[38] each report cases of hemangiomas of the pinna extending medially to encroach upon the external auditory canal.

Benesi[39] reported a case that showed a tumescence of the left external auditory canal which after biopsy and microscopical examination proved to be metastatic hypernephroma. A primary lesion of the left kidney was found later. The patient died of the primary disease. Such a metastasis to the external auditory canal was most unusual.

Secondary invasion of the external auditory canal by neoplasms located elsewhere convey a poor prognosis. This is true even though the original lesion has been eradicated. Invasion of bone of the external auditory canal wall carries even a worse prognosis. Eradication of such an extension demands wide surgical excision and should be undertaken only if the primary neoplasm can be removed with it *en bloc* at the same operation.

E. TRAUMA TO THE EXTERNAL AUDITORY CANAL.

1. WAR INJURIES.

During World War II many traumatic lesions of the external auditory canal were noted and for the most part were associated with other more severe injuries. Blast, shrapnel, bullet or stab wounds invariably caused such severe damage about the head and neck that life itself was endangered. Damage to these other structures demanded treatment first. Only after the patient's life was assured, sometimes after many months of treatment, would the otolaryngologist be in a position to treat these traumatic lesions of the external auditory canal which consisted chiefly of strictures, stenoses, atresias, draining sinuses, loose fragments of bone and foreign bodies of all types.

The treatment of the external auditory canal lesion was planned in advance so that other disease in the neighborhood, for example, mastoiditis and/or splintered bone, could be adequately eradicated at the time of surgery.

CASE 9.—The following injury to a combat soldier's external auditory canal from a bullet wound is typical. The soldier had been shot by a bullet through the left ear. There was a creasing of the mastoid process gouging out a small amount of bone. At exit the bullet fractured the ascending ramus of the mandible. After first aid treatment the patient was evacuated to a base hospital. There the treatment was directed at restoring the shattered mandible and face.

Fig. 7, Case 10.—Six weeks after revision mastoidectomy had been performed for local bullet injury to the left pinna, external auditory canal and mastoid. The facial nerve had been previously anastomosed with the eleventh cranial nerve. Note incision for sliding graft. The mastoid cavity eventually filled with healthy granulation tissue, and the external auditory canal and meatus sealed off completely, which was desirable because of the localized area of pachymeningitis in the middle fossa.

After many months of therapy, the injury to the left external auditory canal which consisted of a complete atresia was treated. Repair of the atresia was done in the same manner as in Case 7 (Fig. 2) except for using a postauricular incision with a Koerner plastic meatal flap. The atresia was membranous and fibrous. The tympanic membrane was intact. The result was satisfactory.

Wounds of sufficient intensity to destroy the bony as well as the membranous canal walls must of necessity be severe enough to damage adjacent structures.

Case 10.—M. K., male, received a bullet wound while in combat April, 1945. The bullet entered the glabella and exited in the region of the left mastoid. He was unconscious for a period of nine days. After evacuation to the interior weeks later, he was found to have a left otorrhea, complete loss of left cochlear and labyrinthian function, left peripheral facial paralysis and partial loss of the left pinna and auditory canal. At operation the splintered bone was removed and since the proximal end of the severed facial nerve could not be found, an anasto-

mosis with the eleventh cranial nerve was performed. Following this he was discharged from the Army. In January, 1947, he was admitted to the hospital complaining of pain and foul otorrhea of the left ear. Examination at this time showed that the left pinna and external auditory canal were deformed. There was foul pus discharging through the deformed meatus. The posterior auricular incision was partially healed. There was a facial asymmetry. X-ray films showed a complete absence of the left mastoid. The mastoid cavity was reopened through a posterior auricular incision in March, 1947. There was a complete absence of the bony external auditory canal with a partial stricture of the membranous portion. Little bone removal had to be done at this operation because of the extensive previous operative procedure. There was a 2 by 3-cm localized area of pachymeningitis where the dura projected into the middle ear cavtiy. This was not disturbed. A Koerner flap was created in the external auditory meatus and the posterior auricular incision was closed by using a sliding graft from over the parietal area (Fig. 7). This patient's ear later healed with complete occlusion of the external auditory canal and meatus. Such healing was desirable because of the localized area of pachymeningitis. Although the patient had a deformed pinna, the ear became dry and safe.

2. FRACTURES.

Fracture of the bony external auditory canal is usually secondary to a severe injury sustained elsewhere in the skull or facial bones.

a. *Basal Skull Fractures.* Patients with fractures of the base of the skull often die immediately or soon after the injury, hence there exists the possibility that many fractures of the bony external canal wall are entirely overlooked. The microscopical sections that Keleman[40] presents verify this statement. The three main types of fractures involving the petrous portion of the temporal bone are the longitudinal, the transverse and their combinations. Grove[41] has pointed out that the longitudinal fracture is the most common type associated with head injury and noted that some of these patients leaked spinal fluid from the external auditory canal which was indicative of a tear into the subarachnoid space.

The patient with basal longitudinal fracture of the petrous pyramid may be comatose. If the external auditory canal has been fractured and the subarachnoid space opened, there will be a blood-tinged pulsating fluid in the canal. The canal wall will be swollen, edematous and ecchymotic. Extensive manipulation in such an ear is contra-indicated. The treatment is directed at saving life and preventing meningitis. Upon recovery the bony external auditory canal may show evidences of the fracture superiorly or posteriorly. The fractured area may be raised and give a door-step appearance that is even discernible to the region of the bony annulus tympanicus. Other evidences of the severity of the original injury may be evident

as facial palsy, deafness, disturbance of labyrinthian function and post-traumatic head syndrome.

Transverse fractures of the petrous bone as a rule are not associated with fracture of the external bony canal.

A combination of the transverse and longitudinal fractures are of such severe nature that death usually ensues.

b. *Fractures of the Facial Bones.* Franctures of the mandible associated with secondary fracture of the bony external auditory canal are not frequently encountered. Force applied to the mandible, particularly in a backward direction, may cause a snapping of the condyle at its neck. However, if the violence is applied in such a manner that there is a dislocation of the mandible posteriorly there may also be a comminution of the anterior part of the tympanic bone. On examination of the canal the skin may appear to be lacerated and bleeding. There is deformity from before backwards. The dislocation may be associated with fractures of other portions of the mandible so that treatment is primarily concerned with the fractured mandible. The bony external auditory canal receives secondary consideration to prevent stricture.

Direct trauma causing fracture of the root of the zygoma or of the squama of the temporal bone may be associated with fracture of the bony external auditory canal. Such injuries are usually accompanied by large areas of contusion and laceration of soft tissue.

3. BURNS OF THE EXTERNAL AUDITORY CANAL.

The most common burn of this type was seen during the war in shipyard workers. While welding, hot slag dropped directly into the external auditory canal in some of the workers. If the molten material happened to literally "freeze" in one spot, there resulted a severe deep burn of the canal without injury to the tympanic membrane. On the other hand if the slag traveled along the canal wall, there resulted a burn that was large, and in some patients a perforation of the tympanic membrane resulted. All treatment was conservative in the form of soothing lotions and measures to prevent secondary infection. In spite of these measures the burns often proved recalcitrant to therapy.

4. FOREIGN BODIES IN THE EXTERNAL AUDITORY CANAL.

Foreign bodies in the external auditory canal usually occur in children. Of the 16,375 eye, ear, nose and throat cases reviewed at the San Francisco City and County Hospital, nine cases of foreign body in the external auditory canal were noted. The foreign bodies included pebbles, beans, glass beads and art gum erasers. All foreign bodies were removed under general anesthesia. Removal of the foreign body in one case was associated with perforation of the tympanic membrane.

When hearing aid impressions of the external auditory canal were taken in plaster,[42] foreign body resulted occasionally due to fragmentation.

III. Lesions of the External Auditory Canal Due to Developmental Disturbances.

A. ATRESIA.

The embryological development of the ear may be associated with unknown factors that lead to deformity. The tubercles of the first pharyngeal cleft that are the anlage of the outer ear may not fuse or develop properly resulting in a deformed, misplaced or even a partially absent pinna. Associated with such a deformity there may be an atresia of the external auditory canal. The surface epithelium forming the external auditory canal may fail to develop properly and/or coincident failure of development of the tympanic ring results in deformity of the canal. It may be in the form of atresia which is bony or membranous or both. Atresias are usually associated with developmental disturbances of the middle ear and its contents including the tympanic membrane. They are less often accompanied by deformities of the inner ear. Congenital facial palsy can be present also.

According to Fraser,[43] males are more frequently affected than females. The deformity occurs more frequently on the right side than on the left. Surgical correction should be carried out if the deformity is associated with otorrhea and mastoiditis regardless of whether the deformity is bilateral or unilateral. He further states that unilateral atresias should not be corrected per se if hearing is good in the unaffected ear.

CASE 11.—J. B., male, aged five years, was born with partial absence of the left pinna and external auditory meatus, and a left facial paralysis. Examination showed normal structures of the right ear. The left pinna was absent except for a small lobe inferiorly and a tit-like nubbin posteriorly both containing cartilage. There was an invagination that suggested a rudimentary external auditory canal although no drum could be ascertained as being present. Another small sinus tract was present in the region where the tragus would normally have been located. It admitted a probe for one quarter inch. A slight conduction hearing loss was present in the left ear. A 512 steel tuning fork showed that its sound was referred to the left ear. Turning tests for labyrinthian response were normal. X-ray examinations of the left ear and mastoid showed: partial absence of the lateral bony external auditory canal, a slit-like opening through it into the middle ear, a large air space for the middle ear with ossicles lying in this cavity, widening of the bony portion of the eustachian tube and hypotympanum, a normal appearing labyrinth and an underdeveloped mastoid antrum.

Comment. No therapy was recommended for this patient because the right ear was normal. At a later date plastic repair of the pinna is to be considered.

B. TUMORS.

Congenital disturbances of development resulting in lesions that appear to be tumors of the external auditory canal are the rarest lesions encountered in this area.

A search of the literature revealed patients with the following types of lesions:

1. TEETH.

In 1936 Marshall[44] reported that he removed two teeth from the external auditory canal of a patient aged 15 years. They were covered by a thin membrane. The hearing was normal and tympanic membrane was intact. Marshall further quotes Becca of Argentina as also having removed a tooth from the external auditory canal of a male patient.

2. TERATOID.

Adam and Gilmour[45] presented in a report the case of a female patient who was seen by them with a growth in the right external auditory canal which had been there for many years. Using a posterior auricular incision, a 2.6 by 1.7-cm growth was removed from its attachment at the jaw joint. Microscopically all three germinal layers were represented in it. The authors raised the question as to whether this tumor may not have arisen from adult squamous epithelium although recognizing its teratoid nature.

3. PAROTID GLAND.

In 1926 Tyler[46] reported the case of a male who had a growth removed from the right external auditory canal in 1920. Because of a recurrence of the mass, the tumor was again removed in 1924. The area was then treated with radium on several occasions until the growth disappeared. Pathological studies of the tumor suggested a cylindroma but there was also parotid tissue in the tumor leading Tyler to conclude that the mass was the result of an embryological disturbance of development of the parotid gland.

C. SEBACEOUS CYSTS.

Although in the strictest sense, cysts of the external auditory canal are not due to developmental disturbances, they are empirically placed in this classification since they may conveniently be considered to be of sebaceous gland anomaly or malfunction. By definition these cysts must of necessity occur only in the outer cartilaginous part of the canal since the sebaceous glands occur only in that part. Politzer[15] speaks of the small "retention tumors," the milium of the external auditory canal. Alexander[47] reported a case with a cyst of the external auditory canal which contained cell debris and fatty oily substance. It was small sized and cured by local excision.

SUMMARY

Certain lesions of the external auditory canal are extremely rare yet considered as a group they are not too scarce and various individual conditions are encountered with enough regularity by the otologist to keep his interest aroused. The present discussion has included the report of 11 cases. A convenient method of classification of lesions of the external auditory canal is to list those diseases arising in the canal, those that involve the canal secondarily by extension, encroachment and contact, and lastly to note the lesions due to disturbances of development. If such a method is used, diagnosis and treatment are made easier.

384 Post Street.

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XCII

POSTNATAL GROWTH AND ADULT STRUCTURE OF THE OTIC (ENDOLYMPHATIC) SAC

THEODORE H. BAST, PH.D.

MADISON, WIS.

AND

BARRY J. ANSON, PH.D.

CHICAGO, ILL.

It is now a well-known observation that the internal ear, with the exception of the otic sac, reaches its adult proportions and content at about midterm or at the time when the otic capsule ossifies. On the other hand the otic sac, the only part of the otic labyrinth which lies outside the petrous bone and between the layers of the dura, continues to enlarge until adulthood.

In the human fetus up to midterm, the otic sac is small and the duct and sac lie in a fairly straight line almost parallel to the crus commune (Fig. 1A). The sac partly overlies the sigmoid sinus into which the rich venous plexus surrounding the caudo-medial surfaces of the sac drains. The close relationship between the otic sac, sigmoid sinus, and posterior semicircular canal are well illustrated in Fig. 1A.

During the latter half of fetal life the posterior cranial fossa begins to enlarge and the sigmoid sinus becomes more and more

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Contribution from Department of Anatomy of the University of Wisconsin and the Department of Anatomy of Northwestern University Medical School (contribution no. 546 from the latter).

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For many of the laboratory procedures and the reconstructions on which this report is based the authors are indebted to Dr. Donald Watzke, whose research was done in fulfillment of part of his postdoctoral training in otology at the University of Wisconsin; for the measurements on the endolymphatic fovea they are indebted to Richard A. Davis, George J. Dennis Fellow at Northwestern University Medical School.

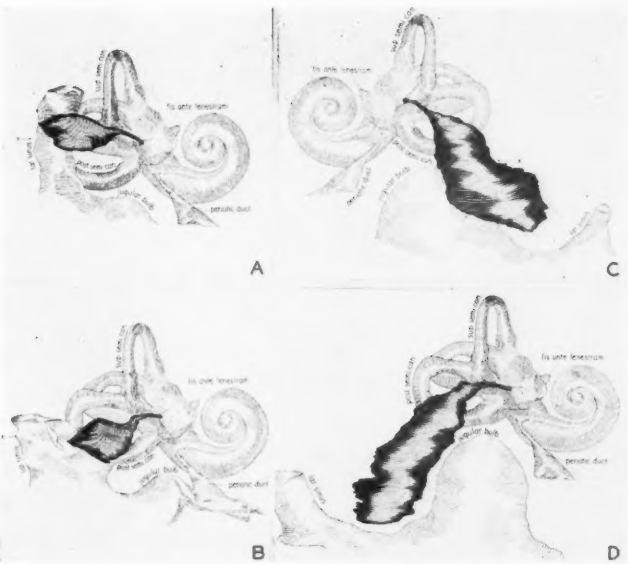

Fig. 1 *A, B, C* and *D.*—Drawings of models of human internal ears reconstructed at an enlargement of 20 diameters and reduced in reproduction to X 2½. The otic sac and that portion of the otic duct not surrounded by periotic tissue is represented by heavy shading. The rest of each drawing indicated by finer shading represents the periotic labyrinth and the lateral (sigmoid) sinus.

A. Wis. series. E 39L, sl. 16, sec. 6, 150 mm., fetus age 18 3/7 weeks.

B. Wis series. E 68L, sl. 11, sec. 2, 310 mm., fetus age 34 weeks.

C. Wis. series. P 24R, sl. 470, female, age 38 years.

D. Wis series. Cad. sl. 89, sec. 2, male, age 65 years.

removed from its close proximity to the posterior semicircular canal. In this process of growth the otic sac is drawn more caudalward within the expanding dura. This results in a decided bend in the otic duct at its junction with the sac (Fig. 1B). This bend persists during postnatal life so that the long axis of the sac forms an angle of from 45 to 60 degrees with that of the otic duct, and the sac comes to overlie the arch of the posterior semicircular canal instead of the crus commune (Figs. 1-B to 1-D).

During postnatal growth the posterior cranial fossa continues to expand and with it the sigmoid sinus is further and further removed from its earlier close relationship to the posterior semicircular canal. The otic sac which lies within the expanding dura must also enlarge. In this way the caudo-medial part of the otic sac together with its vascular subepithelial plexus always maintains a fairly close relationship with the sigmoid sinus. In order to maintain this relationship the sac has to increase threefold or more in length and its width is also correspondingly increased (Figs. 1A to 1D).

Due to the periosteal growth of bone around the original otic capsule the proximal end of the otic sac comes to lie within the slit-like opening of the aquaeductus vestibuli underneath a shelf of periosteal bone (Fig. 2A). The rest of the otic sac (the otic sac proper which is housed between the layers of the dura) overlies a depression in the petrous bone which extends from the slit-like opening of the aquaeductus vestibuli caudally to the impression of the sigmoid sinus (Fig. 2A). The length of this impression, which varies in different individuals, indicates the length of the otic sac. Figure 1-D shows an otic sac which measured 15 mm in length or three times that of fetal sacs shown in Fig. 1A and 1 B. Judging from measurements of the impression in some adult petrous bones recently measured the sac may attain the length of 20 mm.

SIGNIFICANCE OF THE ENLARGED ADULT OTIC SAC

What is the meaning of this structure which has continued to enlarge long after the rest of the internal ear has reached its maximum size? One might think that the sac was passively stretched with the growing dura in which it was imprisoned and as such had no functional significance, as many of the older investigators supposed.

Even though the great size of the otic sac has not been commonly known its function has interested investigators.

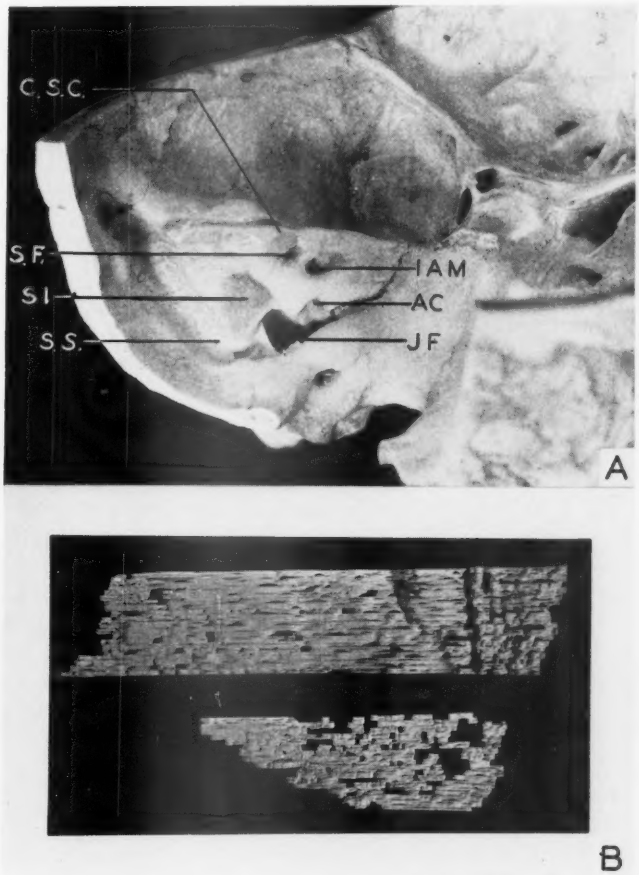

Fig. 2*A*.—Photograph of the posterior aspect of the petrous bone. *C.S.G.*—Crest of superior semicircular canal. *S.F.*—Subarcuate fossa. *S.I.*—Impression produced by otic sac. S.S.—Sigmoid (lateral) sinus. *I.A.M.*—Internal auditory meatus. *A.C.*—Opening of the cochlear aqueduct. *J.F.*—Jugular fasso.

Fig. 2*B*.—Two models reconstructed from serial sections of otic sac shown in Fig. 1*D*. The upper of the two models is from the caudal tip of the otic sac. The holes in these models represent the places where the epithelial sac was interrupted and replaced by connective tissue. Histological sections of this otic sac are shown in Fig. 4*A* and *B*.

A detailed account of the historic opinions regarding the function of the otic sac will not be given here. They have been reviewed by Dr. S. R. Guild[12] in his classical paper on the circulation of the endolymph. Among the more important of the old students of the ear are Boettcher,[9,10] who first described the otic duct leading to the otic sac. He believed that the sac produced the otic fluid which flowed through the otic duct to the otic labyrinth. Rüdinger and others (quoted by Guild) believed there were open communications between the sac and the surrounding lymphatics and noted definitely circumscribed gaps in the fundic end of the sac through which, it was believed, fluid could escape. Thus on somewhat erroneous evidence they believed the fluid flowed from the labyrinth to the sac. Many of the other older investigators considered the otic sac an atrophic portion of the internal ear and as such was non functioning. Guild[12] on experimental evidence showed for the first time that otic fluid moved from the labyrinth to the sac. Guild[11] also noted the rugosities which Boettcher[9, 10] and Sterzi[18] had described. These rugosities and the active appearing epithelium have since been varified by Anson and Nesselrod,[3] Anson and Wilson,[4] Bast, Anson and Gardner,[8] and Bast and Anson.[7] Such observations seem to have strengthened the view that the otic sac is a resorptive mechanism for the otic fluid. Portmann[16] employed this concept in the treatment of vertigo by opening the otic sac to permit drainage of the otic fluid. He reported some favorable results. Hallpike and Cairns[13] reported the absence of the normal vascularity around the otic sac in two cases of Meniere's disease which showed a generalized distension of the otic labyrinth. In order to determine whether the lack of resorption by the otic sac is the cause of the increased fluid pressure in the otic labyrinth, known as hydrops, Lindsay[14] reported that obliteration of the otic sac in monkeys did not seem to alter the normal volume of otic fluid. His animals were sacrified about three months after operation which may have been too short a period for appreciable changes to take place.

Several of the older authors, quoted by Guild, suggested that the otic duct and sac might regulate the pressure of the otic fluid. This view was recently presented by Secretan[17] who considered the loose subepithelial tissue of the sac as a mechanism which permitted expansion and of the otic sac and thus regulated the pressure of the otic fluid in the labyrinth. Alteration of this loose tissue into a dense tissue would prevent distension of the sac and result in a loss

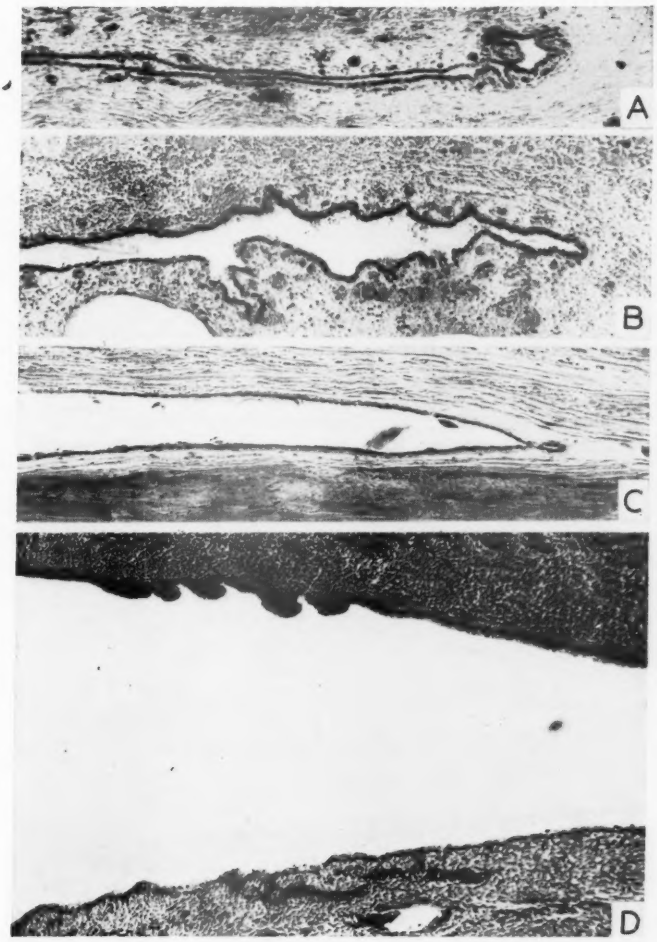

Fig. 3 *A, B, C* and *D.*—Sections through otic sacs. Magnification X 116.

A. The section is taken at the level shown by the *X* in Fig. 1*A.* Wis. series. E 39L, sl. 16, sec. 6, 150 mm., fetus age 18 3/7 weeks.

B. A section from the level shown by *X* in Fig. 1*B.* It shows the medial rugose edge of the sac. Wis. series. E 68L, sl. 11, sec. 2, 310 mm., fetus age 34 weeks.

C. A section through the lateral part of the otic sac at the level shown by the *X* in Fig. 1*C.* Wis. series. P 24R, sl. 470, female, age 38 years.

D. Section through the medial part of an otic sac which was large and dilated. Note the dense subepithelial connective tissue. Wis. series. P 10L, sl. 425, male, age 62 years.

of pressure regulation within the otic labyrinth as in Ménière's disease.

HISTORICAL STRUCTURE OF THE ADULT HUMAN OTIC SAC AND THEIR FUNCTIONAL IMPLICATIONS

The adult otic sac may be divided into: (1) A proximal part, which lies within the slit-like expanded end of the aquaeductus vestibuli, and (2) the otic sac proper which lies within the dura outside of the otic capsule. The distal projection of the otic sac described by Macklin,[15] Anson,[1, 2] and Bast[6] for human fetuses is absent in the adult. It has no doubt been absorbed by the enlarging sac proper.

For Histological and functional considerations the sac proper should be further subdivided into a medio-caudal part which as stated above lies close to the sigmoid sinus, and a lateral part. As described by Bast[5] the medio-caudal part of the otic sac is surrounded by rich venous plexus which drains into the adjacent sigmoid sinus. At all stages of development this part of the otic sac lies in close relationship to the sigmoid sinus (Fig. 1A-1D).

THE SUBEPITHELIAL CONNECTIVE TISSUE

The subepithelial connective tissue surrounding the vascular plexus of the medio-caudal part of the sac is very loose and fine fibered (Fig. 4B, 4C, and 5B). Outside of this loose vascular connective tissue is the dense fiberous tissue characteristic of the dura. Around the lateral part of the sac the loose vascular tissue is absent and the dense dural tissue forms the subepithelial bed (Fig. 3C and 4A). It should be noted that Fig. 4A is from the lateral and Fig. 4B from the medial part of the same histological section through an adult otic sac. This loose subepithelial connective tissue was noted by Hallpike and Cairns[13] and by Secretan[17] but its specific localization was not recorded. Hallpike and Cairns[13] noted the absence of this loose vascular connective tissue in two cases of Meniere's disease which showed generalized distension of the otic system. In a distended sac (Fig. 3D) the absence of this loose connective tissue can be varified. Unfortunately the clinical history in this case is not available. Secretan[17] considered this loose tissue as a mechanism to permit expansion of the sac. If so why is it not present in the lateral part?

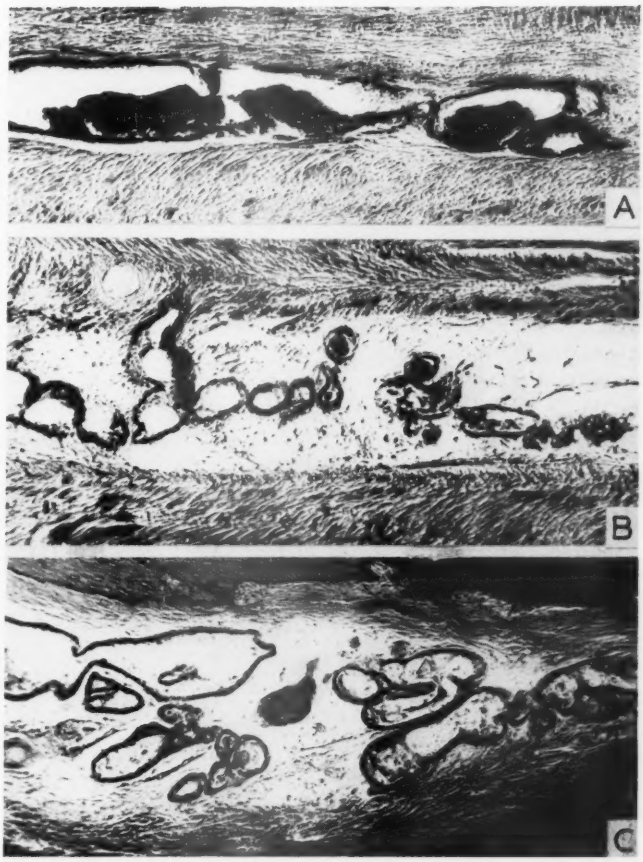

Fig. 4 *A, B, C.*—Sections through otic sacs which were interrupted. Magnification X 116. Figures *A* and *B* are taken from the middle portion of the otic sac shown in Figure 1*D*. Wis. series. Cad. sl. 89, sec. 2, male, 65 years. Figure *A* is taken from the lateral part of the sac and shows the dense subepithelial connective tissue. The sac contains dense staining debris. Figure *B* taken from the medial part of the sac shows many interruptions and the subepithelial connective tissue is very loose and contains a rich vascular plexus. Figure *C* is similar to B, but from a younger individual. Note the abundant stainable debris in the lumen of the sac. Wis. series. P 25R, sl. 387, male, age 17 years.

THE LUMEN OF THE SAC

The lumina of the otic sacs are as a general rule slit-like, although distended sacs, as noted above, are found. Along the lateral part of the sac the walls are usually smooth (Fig. 3C and 5C) but in the proximal and the medial part of the sac the epithelial walls are as a rule thrown into folds or rugae (Bast and Anson[7]). The subepithelial connective tissue in the rugose regions is of the loose vascular type (Fig. 3A and B, 4B and C and 5B). Because of these rugae the lumen becomes markedly distorted. In some cases the epithelia of opposing rugae meet and fuse (Fig. 5A). The epithelium of such areas may then distintegrate and be replaced by loose vascular connective tissue (Fig. 5B). If this process of interruption becomes extensive the lumen becomes a plexus of spaces with a continuous lining of epithelium, surrounded by the loose vascular connective tissue. Such sacs in histological section appear as a series of pockets (Figs. 4B, 4C and 5D) and in reconstructions (Fig. 2B) the interruptions are indicated as holes. In this type of sacs the venous plexus within the loose subepithelial connective comes in close relationship with much larger areas of the epithelial sac than is otherwise possible. It may be that Rüdinger saw such interrupted sacs and regarded the holes as denuded areas of the epithelial sac.

Guild[11] noted cellular debris in the otic sac. Stainable material has also been noted in the adult human. In sacs that are distended (Fig. 3D) little or no stainable material is found, but in the more collapsed or interrupted sacs various types of stainable debris can be seen (Fig. 3B, 3C, 4A, 4B, 4C, 5C and 5D). Such debris may be the more solid material not resorbed through the sac wall.

POLARITY OF THE EPITHELIUM OF THE OTIC SAC

The epithelium, lining the otic sac, varies from a low or flattened to a columnar type. It is usually taller in the rugose or medial part than in the smooth or lateral part of the sac. In either case there is a marked tendency for the nuclei of the epithelial cells to lie nearer the free than the basal margin of the cells (Fig. 5C and 5D). This peripheral location of the nucleus gains significance if one recalls that in simple columnar epithelia, which function as external secreting glands and eliminate their product from the free end of the cell, the nucleus is, as a rule, placed near the basal end of the cell. On the other hand in internal secreting glands and certain

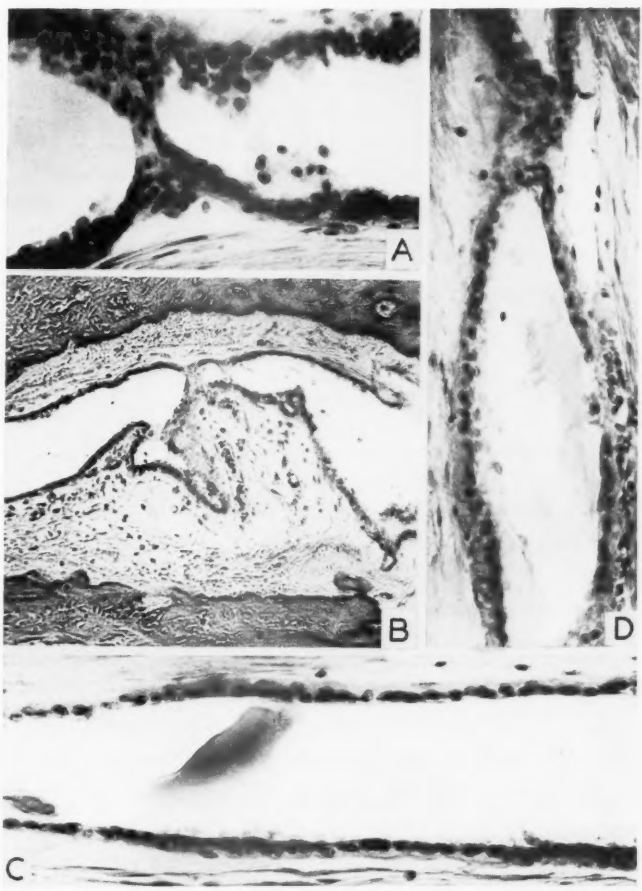

Fig. 5 *A*, *B*, *C* and *D*.—Photomicrographs of portions of the otic sac.

A. Wis. series. P. 37L, sl. 325, female, aged 8 years. X 300. This shows the fusion of the epithelium of two opposing rugae.

B. Northwestern Univ. series. 6.8.'29. Age 3 years. Magnification X 170. This shows the breakdown of the fused epithelia and their replacement by connective tissue. More advanced stages of this process of interrupting the continuity of the epithelial sac is shown in 5D and in 4*A*, *B* and *C*. *C* and *D* show besides the stainable content of the sac the reversed polarity of the epithelium. In both cases the nuclei lie near he free margin of the cell.

C. Wis. series. P. 24R, sl. 470, female, age 38 years. X 300.

D. Wis. series. P. 13L, sl. 450, male, age 62 years. X 300.

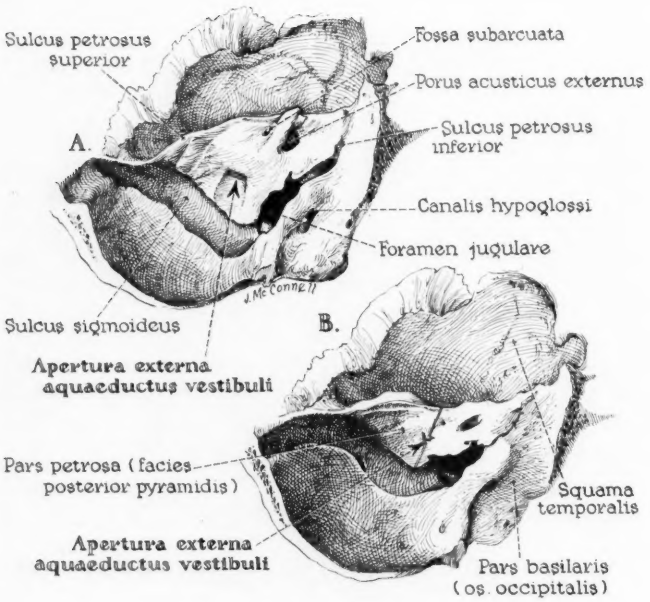

Fig. 6*A*.—Left temporal bone of an adult, seen from the hind. Showing on the posterior surface of the petrous pyramid, the large cranial aperture of the vestibular aqueduct, the shielding ledge of bone, and inferior to the latter, a distinct fovea for the endolymphatic sac.

Directions of measurements are indicated (by arrows) for the distance between the aperture and the superior petrosal sulcus and for that between the aperture and sigmoid sulcus.

Fig. 6*B*.—Left temporal bone, from another adult skull. The aperture is small; a fovea is wanting.

resorptive epithelia where the cells pour their secretion through the basal end, as in the beta cells of the pancreas, or the cells of the thyroid or the ameloblasts of the enamel organ, there is a marked tendency for the nuclei to lie at the opposite pole or free margin of the cell. If such reversal of polarity is of any functional significance then the epithelium of the otic sac should have a resorptive function.

SUMMARY

1. The adult otic labyrinth attains adult proportions at about midterm whereas the otic sac enlarges to three times or more the midterm size in postnatal life.

2. After midterm the otic duct bends so as to permit the medio-caudal part of the otic sac to retain a close relationship with the sigmoid sinus.

3. The medio-caudal part of the sac like the proximal part is usually rugose and is immediately surrounded by a very loose vascular connective tissue whose venous sinuses drain into the sigmoid sinus.

4. Fusion of opposing rugae may result and such fusion spots may be replaced by connective tissue. Thus the sac may become interrupted and its lumen will be converted into a series of connected and epithelium lined channels.

5. The nuclei of the lining epithelium are as a rule located near the free margin of the cells.

6. These anatomical observations tend to support the functional concept that the otic sac is a drainage organ for the otic fluid.

303 East Chicago Ave.

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DISCUSSION

DR. BARRY J. ANSON (Chicago, Ill.): As described by Dr. Bast, the endolymphatic (or otic) sac is the only part of the labyrinthic system of the human ear which has not attained full, or adult dimensions at midterm, that is, the fetus of 183 mm crown rump length. Housed partly within the vestibular aqueduct and partly beyond the orifice of the latter, upon the posterior surface of the petrous pyramid it continues to enlarge even in the adult body. The cochlear and semicircular ducts, the utricle and sacculus, are contained within an osseous, otic capsule whose growth terminates at midterm. The endolymphatic sac on the contrary, continues to grow into early adult years.

As has been established, the complexity and size of the rugosities vary with the subject studied and with portions of an individual endolymphatic sac. The folds vary from the inconspicuous to the well-marked. In some cases they are broad hummocks, as seen in sections; in others the rugae are so tall as to touch the opposite epithelial wall of the sac, or even to fuse with it in such a way as to convert the lumen into a series of epithelial pockets. In some instances the rugae appear villous.

In a selected specimen of an adult skull the internal (cranial) aperture of the vestibular aqueduct is exceptionally large, as is also the exposed portion of the fovea situated inferior to the aperture for lodgement of the endolymphatic sac (Fig. 6*A*). As is regularly the case, a ledge of bone guards the orifice of the aqueduct; therefore, the latter opens into the cranial cavity through an infundibular enlargement. In this specimen the fovea is prominent (through the possession of distinctly elevated margins) and elongate (reaching the sigmoid sulcus). In life the edolymphatic sac doubtless overlay the sigmoid sinus. The subarcuate fossa is small, as is regularly the case in the adult. In a second specimen of the adult skull, by way of contrast, the cranial orifice of the vestibular aqueduct is exceeptionally small (Fig. 6*B*); the ledge of bone is correspondingly narrow; a fovea for the sac is wanting.

In recording the form and size of the endolymphatic sac, a series of 200 temporal bones, obtained from the Laboratories at Northwestern University Medical School and the University of Wisconsin, were employed. Of the 200 temporal bones measured it was found that in 139 specimens, or 70 per cent, a definite impression of fossa made by the endolymphatic sac was present.

In this series the distance from the sulcus of the superior petrosal sinus downward to the bony ledge (superior limit of the intracranial part of the fossa) varied from 4 mm to 13 mm. The measurements from the sigmoid sinus upward to the bony aperture of the vestibular aqueduct ranged between the extremes of 3 mm and 22 mm, in 200 temporal bones.

In form, the fovea for the endolymphatic sac was usually oval; however, many variations occurred.

Dr. John R. Lindsay: I have greatly enjoyed this illustration of this most meticulous and careful study of the extent of the otic sac. My interest in this structure became rather stimulated because of its possible relation to not only the treatment of Meniere's disease as advocated by Portmann, but also Hallpik's explanation that interference with the function of this structure might be the cause of hyedrops of the labyrinth.

We studied a number of temporal bones at that time to see if we could agree with Hallpike's idea that this change in the perivascular connective tissue might be interpreted as an explanation for the hydrops. I must confess that we could not quite make those interpretations from our slides. I think later Hallpike has admitted that they find the same change in the perivascular connective tissues in ears that do not have hydrops and on the other hand some that do have hydrops do not have this change, so we wondered if, perhaps, the histological appearance could be entirely reliable. As an example of instances where it has not been reliable, I might point out, for instance, the appearance of the cupula, as we have always seen it in histological sections. We now know as the result of Steinhausen and Dohlman's work that the cupula is an entirely different structure from what we see in histological sections. It appeared to me that if we could in some way do something to disturb the function of this structure we might get more information regarding its function. Therefore, it was the obvious thing to do to destroy the duct and sac in the animal and see what happened. I thought perhaps we were going to produce a hydrops, but I might show a couple of these sections to show what actually did happen.

XCIII

VASOMOTOR LABYRINTHINE ISCHEMIA

JEROME A. HILGER, M.D.

ST. PAUL, MINN.

The eighth cranial nerve with its acoustic and vestibular sensory apparatus is prone to pathologic change and disability far beyond any of the other cranial nerves with the possible exception of the second. Those numerous disorders classified for decades as primary eighth nerve neuritis are subject to critical re-evaluation. Why should the eighth nerve exhibit a peculiar affinity for primary neural disorders, nutritional or otherwise, that uncommonly beset other of the cranial nerves? It is not reasonable that it should. It is certain that it does not. The sensory end apparatus of the eighth nerve is the differential feature between it and the other cranial nerves. It is there one must look for the source of the commonplace perceptive, acoustic and vestibular disturbances encountered in everyday otologic experience.

The sensory epithelia and primary neurons of the eighth nerve have been invested with magnificent protection against infection and direct trauma. The price that is paid for the protective encapsulation of this end organ is the hazard of end circulation. The vital intralabyrinthine structures depend for the integrity of their function and structure upon end arteries—there is no collateral circulation.

ANATOMY

Features in the vascular anatomy of the labyrinth which have important bearing on clinical signs must be placed in mind.[1] The labyrinthine artery has two main manners of division:

I. Labyrinthine artery (Fig. 1)
   a. Anterior vestibular artery
   b. Common cochlear artery
      1. vestibulo-cochlear artery
      2. cochlear artery proper

II. Labyrinthine artery (Fig. 2)
   a. Vestibular artery
   b. Cochlear artery

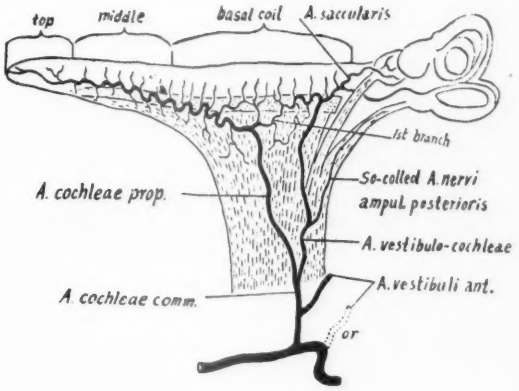


Fig. 1.—Arterial distribution to the labyrinth. (from Nabeya)

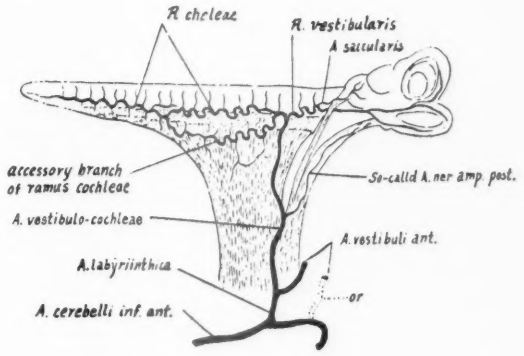


Fig. 2.—Arterial distribution to the labyrinth. (from Nabeya)

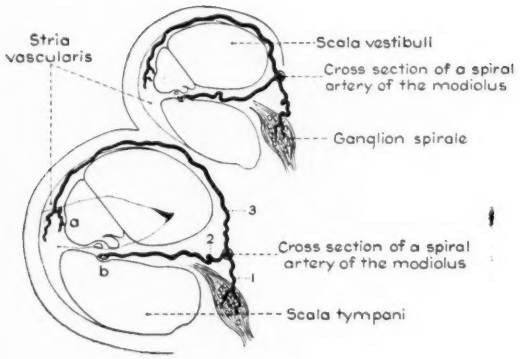


Fig. 3.—Ramifications of the cochlear artery.

The main portion of the vascular system of the labyrinth courses on the endosteal wall of the labyrinth while the terminal arteriolar-capillary-venular portion distributes on the membranous labyrinth. The blood vessels of the membranous labyrinth are not related to those of the bony capsule; hence, there is no collateral through the blood supply of the capsule.

In the cochlea the terminal arterial and arteriolar divisions are of three types from the spiral arterial stem (Fig. 3):

1. The serial twigs to the spiral ganglion cells.
2. The radiating arterial branches on the lamina spiralis.
3. The radiating arterial branches on the scala vestibuli which supply the stria vascularis and the spiral ligament. In the stria the capillaries lie in the epithelium of the cochlear duct.

In the vestibule and the semicircular canals the terminal vessels form a fine network on the macula utriculi, the macula saculi, and the ampullary crests. In addition, in the vestibular nerve there is formed from vestibular arterial branches a peculiar arteriolar network which supplies the vestibular ganglion cells. This capillary network on the vestibular ganglion cells is more dense than that on the cochlear ganglion cells, and may afford the reason for the greater frequency of ischemic neural tinnitus and hearing impairment in the latter as contrasted with ischemic neural vertigo in the former.

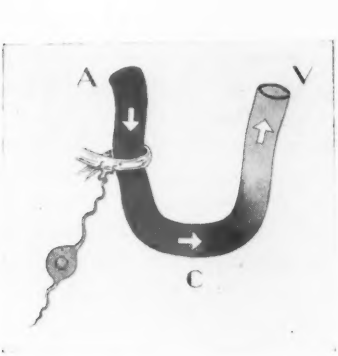


Fig. 4.—The functional vascular level—the arteriolar-capillary-venular unit.

The principal venous channels of the membranous labyrinth course along the endosteal labyrinthine wall and drain through two channels.

1. The vein of the cochlear canaliculus collecting from the cochlea and a portion of the vestibule.

2. The vein of the vestibular aqueduct which collects from the remainder of the vestibule and the semicircular canals.

PATHOPHYSIOLOGY

The tissues and organs of the body which unfortunately lack collateral circulation have common clinical problems resulting from a disturbance in end-circulation. The disturbed vascular physiology which produces peripheral end-arteriolar ischemia is of the utmost importance to the rheumatologist, the cardiologist, the neurologist, the occulist, and the otologist—to mention only a few special fields with this common interest. The otologist occupies a signal advantage in the study of peripheral vascular disturbance. The neural elements of the labyrinth are extremely sensitive to and expressive of ischemic change. One is permitted clinically to focus down to the arteriolar level in studying functional disturbances of the normal vascular structure. This stage of clinical medicine far precedes the stage of organic vascular obliteration.

The study of the functional behavior of the structurally normal arterial bed is technically more difficult than the pathologic study of the terminal organic change. The functional level of the vascular bed is the arteriolar-capillary-venular unit (Fig. 4). Disturbance begins with spastic narrowing of the arteriolar lumen. The capillary balloons, due to ischemic, hypoxic, endothelial damage and reflux venous tonus (Fig. 5). Transudation between endothelial cells results in extravascular fluid.[2, 3, 4] The latter compresses lymph capillaries and localized stasis is complete. This cycle of events comprises the motor mechanism of wheal formation. That it can be massive and sudden is attested to in the formation of giant urticaria. Where collateral circulation is present the intact neighboring vascular unit clears the field of its edema. Where there is no collateral the arteriolar spasm must relax and normal circulation be re-established through the involved vascular unit before the extravascular fluid will restore to the circulation. This is the unhappy circumstance in the labyrinthine vascular terminals.

SYNDROMES

With this neurovascular mechanism in mind one can project and predict the symptoms and findings resulting from this vasospastic cycle in the end vessel arborizations of the labyrinthine artery. The neural changes will vary in degree, extent and permanent residua as the ischemia and transudation vary in degree, extent and duration.

Spasm in the vascular terminals supplying cochlear ganglion cells (Fig. 6) must result in discrete symptoms. Tinnitus and hearing impairment will be specific for the cell areas involved. The loss will be purely perceptive. Any frequency zone may be affected. The fact that the drop is more frequent in the high tones must be dependent on anatomic features. So also must the frequent association of vertigo with high tinnitus and high frequency decline be due to arteriolar spasm in the vestibulo-cochlear segmental periphery. No sensation of aural pressure will accompany ischemia in the ganglion segments. There will be no diplacusis. Loudness recruitment may be absent as noted by Dix, Hallpike, and Hood[5] in their patients with an intact end organ, but with degeneration of the neural elements resulting from acoustic neuroma. Though this aspect needs further study, it may open an avenue for differentiation of vascular changes in specific areas within the labyrinth.

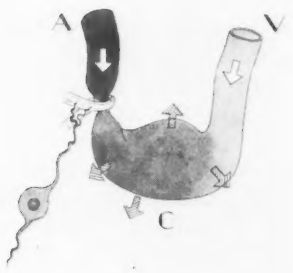


Fig. 5.—Arteriolar spasm and its sequelae.

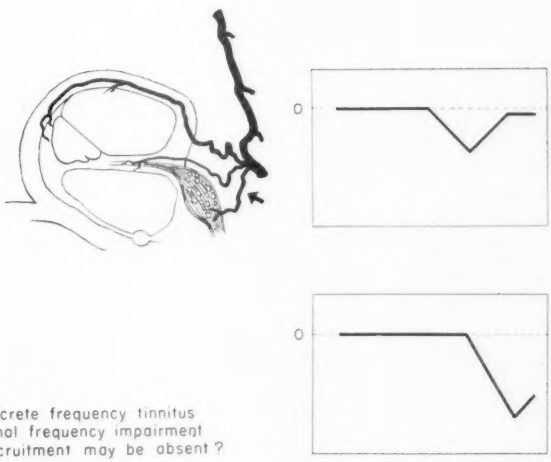
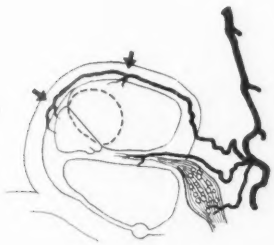


Fig. 6.—Ischemia of the cochlear ganglion cells (catarrhal deafness, eighth nerve neuritis, etc.).

The arterial branches over the scala vestibuli to the stria vascularis are the source of endolymph excess when affected (Fig. 7). Endolymphatic hypertension due to this transudative or secretory mechanism must vary tremendously in degree. There is evidence to support thinking that perilymphatic hypertension due to similar transudation into the scala vestibuli occurs in some instances instead of endolymphatic excess.[6] Symptoms in either instance will result from distortion of sensory epithelia in acoustic and vestibular segments. Vertigo which occurs at the inception of lymphatic hypertension will tend to subside as the status quo of epithelial pressure and distortion is maintained. Tinnitus will not be specific. It will cover a broad tonal range. Sound perception in instances of moderate tension increase will be first distorted on a broad front even before a pure tone audiometric drop will be demonstrable. Aural pressure will be experienced. The false concept of tubal impairment due to the patient's complaint of sound distortion and pressure sensation will often, in this phase, encourage the loss of valuable therapeutic time in purposeless tubal inflationary piddling. When endo- or perilymphatic hypertension is more pronounced conductive impairment will increase. Bone conduction will be reduced. Increased lymphatic tension favors rather than discourages the conduction of high frequencies due to increased rigidity. High tones will tend to remain at a better level or even be increased—the so-called paradoxical upper limit of Dederding.[7] This is only true if there is no parallel ischemia in the ganglion cell area. Diplacusis will be demonstrable with pitch elevation present in the affected ear. Loudness recruitment will be present. Distortion and recruitment will combine to make the reception of loud sound unpleasant and even painful.

Ischemia in macula or ampullary crest (Fig. 8) may not be symptomatic until normal physiologic stimulus of movement in the plane or planes involved fails to provide the usual response from the sensory terminals. Vertigo will be experienced only on movement in the discrete axis involved.

The arteriolar network to the vestibular ganglion cells is rich in supportive communication as compared with that to the cochlear ganglion area. Vasospasm (Fig. 9) is less likely to produce discrete ischemic damage. When sufficient segments are involved cell damage will produce, as with tinnitus from a cochlear ganglion cell in-



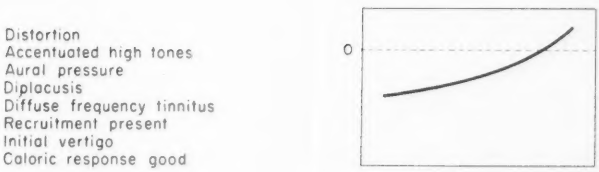
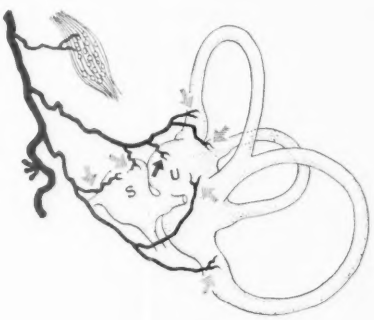
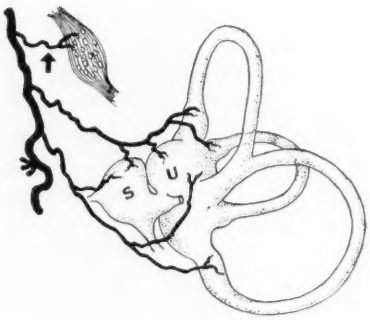


Fig. 7.—Ischemia in the stria vascularis with formation of endo- and/or perilymphatic hypertension (Meniere's disease, endolymphatic hydrops with vertigo, labyrinthosis, etc.; or, if endolymphatic hypertension is not transmitted through utricular valve—endolymphatic hydrops without vertigo).



Vertigo (persistent with movement in the affected plane)
Caloric response impaired (if end-plate damage is severe and test is discriminative)

Fig. 8.—Ischemia in macula or ampullary crest (pseudo-Meniere's disease).



Vertigo (not confined to a discrete plane of movement. Often persistent without movement)
Caloric response impaired (if neural damage is sufficient)

Fig. 9.—Ischemia in vestibular ganglion cells (pseudo-Meniere's disease).

jury, a continuous neural volley centralward which will give complaint of persistent vertigo of variable degree, but not dependent on position change for instigation.

Massive or total labyrinthine arteriolar spasm (Fig. 10*A* and 10*B*) is not uncommon. A mixed picture of neural and lymphatic hypertensive change will result. Symptoms and signs will be due to a summation of ischemic damage. One cannot in such instance dissociate one element from the other.

As an apologia for this thesis of discrete labyrinthine phenomena one must only be pleased to remember that each of the projected symptom complexes does occur; that they occur in the daily experience of any otoligist if he analyzes, particularizes and recognizes, and that they can only be explained on the basis of discrete vascular behavior for which all of the anatomic features and the method of disturbed vascular physiology have been well established.

THERAPY

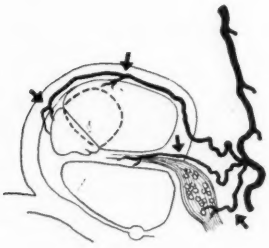
Therapy for the vasomotor labyrinthine crisis aims at correction of existing vasospasm and resorption of transudates (Fig. 11). In

the recurrent state prophylactic therapy discourages vasospasm and restores and maintains the integrity of the ischemically damaged endothelium.

The crisis is neurovascular and demands a neural block. It is less logical to treat it by a generalized vasodilatation since a limited total blood volume can sink into a dilated vascular periphery without special benefit to any region. The most effective method of increasing local blood supply is sympathetic denervation of the part. Local neural block can be maintained by a continuous cervical sympathetic block at the stellate ganglion. One-tenth percent procaine hydrochloride solution dripped through an indwelling polythene tube is effective. This technique avoids the pendulum swing of block and reconstriction which sometimes follows intermittent procaine injection of the stellate.

As an alternative and more readily available method, procaine hydrochloride is very effective as a peripheral neural blocking agent if used in diluted one-tenth or two-tenths percent solution as a continuous intravenous infusion. It is quite selective in its action for the vasospastic bed. It appears to pool in the region of transudate and maintain action even after the infusion has been terminated. It is easy to handle and has a minimum of disagreeable or dangerous side affects. As a discrete intravenous injection in a more concentrated dose, it was advocated by Lewy[8] some years ago for the treatment of tinnitus aurium. The rationale for its use was not understood. It was based on Barany's accidental observation of the occasional relief of tinnitus after procaine injection into the inferior turbinate during intranasal surgery.

The combination in one infusion of a neural blocking agent and a peripheral vasodilator suggests a most intriguing therapeutic possibility. As noted heretofore, a dilator which simply increases the general peripheral bed is of limited purpose and in some instances may result in more relative ischemia to the labyrinth. However, a vasodilator which would in addition increase the heart action so the total blood volume would be passed through the increased periphery more often in a given time would approximate the ideal. This is the action of adrenalin.[9,10] Contrary to popular misconception, it is one of the most effective of vasodilators when used in physiologic concentration. In addition it increases heart action—that is the blood volume handled by the heart in a given time—by seventy to



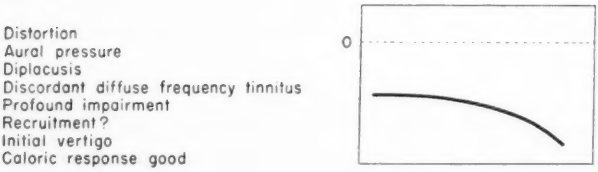


Fig. 10*A*.—Total cochlear ischemia (Meniere's disease, endolymphatics hydrops with vertigo, labyrinthosis, etc.; or, if endolymphatic hypertension is not transmitted through utricular valve—endolymphatic hydrops without vertigo).

ninety percent. The action of adrenalin as a labyrinthine vascular dilator has been demonstrated histologically by Mygind.[11] He spoke of it as paradoxical. Actually it is physiological. Histamine was shown in the same experimental series to constrict the vessels of the stria vascularis. This is a disturbing observation in light of the current proneness to intravenous histamine therapy.

We combine in our intravenous infusion for the neurovascular crisis one to two grams of procaine hydrochloride, one-tenth milligram of adrenalin hydrochloride, and one gram of ascorbic acid to the liter of diluent. The procaine unit[12] of 4 mgm/kgm as a single thirty minute infusion dose is extremely conservative. It is a safe base from which to feel one's way in familiarizing himself with new therapy. A greater total dose in a longer infusion period for more prolonged blocking action is our present custom. In hundreds of injections we have had no untoward evidences of toxicity or sensitivity.

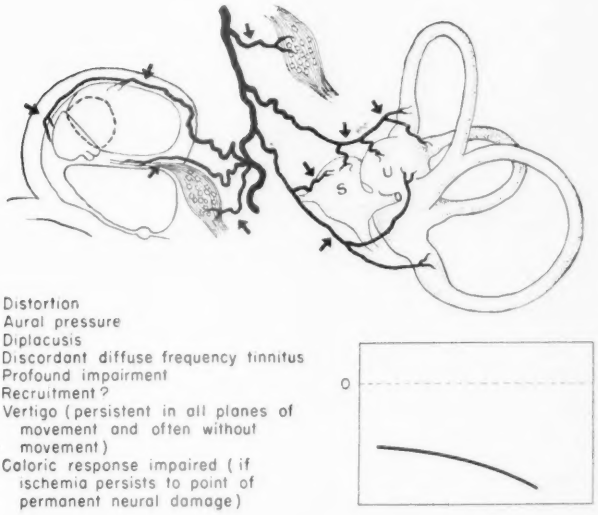


Fig. 10*B*.—Total labyrinthine ischemia (Meniere's disease, endolymphatic hydrops with vertigo, labyrinthosis, etc.)

The vasospastic phase of the labyrinthine vascular crisis is amenable to blockade therapy and the transudate in the vascularized portions of the labyrinth is readily resorbed through the restored vascular bed. Where spasm in the radiating vessels to the stria has been operative the increased perilymph and endolymph presents a different problem of resorption. Indeed where the lymphatic hypertension has become extreme to the degree characterized as hydropic, the potentially resorptive venous capillary loops in the epithelium of the spiral ligament, and immediately beneath the perilymphatic endothelium, and the epithelium of the endolymphatic duct must be visualized as compressed by the tension and relatively useless for resorptive action. Diffusion through the cochlear canaliculus or endolymphatic duct will eventually lower the tension if continual transudation from the arterial side is halted. The situation in endolymphatic or perilymphatic hypertension must be likened to that of glancoma. At best the reduction of severe tension is slow. The "pump action"[13] of a properly functioning, pulsating labyrinthine arteriolar periphery is the most direct influence we can therapeutically exert. Sodium

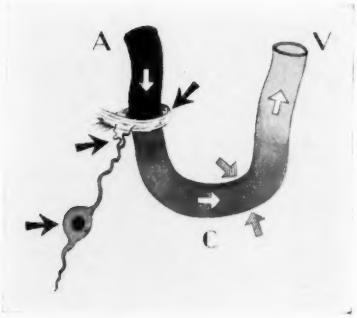


Fig. 11.—Points of therapeutic focus: the smooth muscle arteriolar spincter; the myo-neural junction; the autonomic ganglion cell.

restriction and limitation of fluid intake are only to moderate purpose. Dehydration with the mercurial diuretics and hypertonic glucose is more effective. However, at best the problem of resorption of the lymphatic hypertensive phase of the labyrinthine vascular crisis remains most difficult and unsatisfactory.

Endothelial tissue which has been subjected to recurrent ischemic damage remains in a state of easy transudation. The restoration of damaged endothelium is occupying a forward position in medical research today. At present three therapeutic supplements appear useful; ascorbic acid, thyroid, and the steroids. Ascorbic acid is incorporated with the intravenous blockade therapy for this reason. Steroid therapy will be an important addition as its use becomes better defined.

Many factors combine to produce the initial severe neurovascular disturbance in the labyrinth. Physical environmental extremes and psychomotor factors are chief impetuators. They activate imbalance more readily in periods of autonomic strain, as following systemic illness, at the time of menses or menopause, in periods of fatigue, etc. The vasospastic habit of smoking is an additional important contributing factor. The individual who manifests signs of cranial autonomic irregularity as recurrent vascular headache, cervical myalgia, chronic vasomotor rhinitis, or vasomotor labyrinthine ischemia has continuity in a positive family history of similar dis-

orders. He should be forewarned of the significance of ischemic inner ear symptoms. Insidious degrees of neurovascular change should be sought out by a routine audiometry.

Neurovascular disturbances readily recur. Habituation of disturbed behaviour in one peripheral area is the rule. Continuation of faulty living is a sure road to habituation. Destruction of a labyrinth through recurrent ischemia or surgically as a method of therapy for intractibly recurrent ischemia is unnecessary if initial signs are heeded, first labyrinthine crises rapidly treated, and the patient is intelligently willing and able to modify his way of life thereafter.

Prophylaxis against advancing labyrinthine change through a continuous preventive regime has merit. Drug therapy is, in addition to correction of the manner of living, purposeful. An entire new field of blocking agents, both adrenergic and cholinergic, are under investigation. Some will deserve a place in continuous preventive therapy. Currently nicotinic acid and B-pyridyl-carbinol are useful prophylactic agents as peripheral vasodilators if used in adequate dose at frequent intervals every day. Large doses of ascorbic acid should be added to the daily regimen and thyroid to tolerance. The steroids may replace this program in part. It remains to be seen.

The otologist occupies a prominent position in clinical medicine today. He is not just a temporal bone carpenter or a tubal piddler. Not, that is, if he appreciates the fineness of the labyrinth as a vasomotor barometer and keeps himself abreast of clinical medical advances. The otologist is equipped to control the vascular phenomena of the labyrinth to the patient's best advantage.

SUMMARY

The vascular anatomy of the labyrinth is vital to its function because it is endarterial.

The disturbed physiology of vasospastic change results in primary neuronal ischemia and lymphatic hypertension.

The syndromes of the inner ear which result can be massive and sudden or minute and insidious.

These syndromes comprise a vast segment of otology.

Their understanding and successful management necessitate a background of knowledge of vascular physiology, labyrinthine anatomy and function, and neurovascular therapeutics which lifts otology out of its former narrow limits.

The mechanical and bacterial era of otology is over. We must embrace changing concepts in a changing specialty.

444 Lowry Medical Arts Bldg.

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XCIV

THE PATHOLOGY OF HEREDITARY DEAFNESS

CHARLES E. KINNEY, M.D.

CLEVELAND, OHIO

All of us have had to inform parents that in our opinion their child had very little if any usable hearing. It has been my unpleasant task to do this about 250 times during the past 15 years. I have always assumed an optimistic attitude in such instances and immediately have arranged for these parents to see and talk to one or more of our older students who were good lip readers and who had a fairly good voice. About two years ago, I was consulted by two young parents with their 28 month old son, Richard. I did not make a positive diagnosis at that time but advised that Richard be enrolled in our experimental pre-school class. After observing this boy for about six months and with the help of some additional history, a diagnosis of sub-total hereditary deafness was made. The barrage of logical questions from those parents was heart-rending. Why did this happen to our son? Where is the location of the trouble? In this day of miracle drugs for this and that why haven't you ear doctors done something about this problem? How can this be prevented in our next child? This paper is an attempt to figure out this problem—at least in my own mind. The answer is still not good.

A complete survey of all published volumes of The Cumulative Index Medicus was made and from their titles 42 articles seemed to bear on the subject of Hereditary Deafness. All of these articles were studied including the 10 that were in foreign languages. In doing this, one was definitely impressed by two outstanding conditions. The first was the marked confusion in the use of descriptive terms concerning deafness and hard-of-hearing. The second observation was the paucity of pathological material. For instance, I was unable to find a single human case in which there was an accurate history and acceptable hearing tests combined with pathological study of both the temporal bone and brain. There is not one American case. This is a very shocking condition and I would urge that effort

be put forth to obtain such specimens from cases that might be within our knowledge. Study of such specimens will be more valuable if they are processed in the same manner. Therefore, a central repository for such case histories and specimens would seem to be the answer and I know of no better body than The American Otological Society.

Politzer[1] in his text book devoted considerable space to deafmutism and many succeeding authors add confusion to this misnomer. In working out this problem for Richard's parents, it became necessary to develop a classification of congenital deafness. This was done on a combined etiological and pathological basis using the modern dictionary definitions of congenital, and hereditary deafness.[2] These definitions are respectively: Existing at birth; dependent upon the constitution of the cell or cells which form the starting point of the new individual and; loss, lack, or impairment of the sense of hearing. This classification is as follows:

CONGENITAL DEAFNESS

I. Hereditary
   A. True Mendelian
      1. Dominant
      2. Recessive
   B. Erythroblastosis Fetalis
   C. Secondary to Other Hereditary Traits
II. Malformations of External or Middle Ear
III. Occurrences in Mother During First Trimester of Pregnancy
   A. Rubella or Other Virus Diseases
   B. Toxic Poisoning - Quinine - Alcohol
IV. Intrauterine Accidents
V. Birth Injuries

Therefore, it is obvious but not always appreciated that all cases of hereditary deafness are congenital but not all cases of congenital deafness are hereditary. In the author's opinion any of these types of congenital deafness may be unilateral, bilateral, total, sub-total or partial. We are indebted to Goodhill[3] for having pointed out the

newest types in the above classification, namely; erythroblastosis fetalis. / He presented this before the American Academy of Ophthalmology and Otolaryngology last fall and I expect him to elaborate on the problem here today. It is hoped that the term "mutism" may be dropped forever because these patients' inability to talk without special training is not congenital. Of the 186 cases of bilateral congenital deafness that I have studied, 59 or 32% of them were classified as probably of the true Mendelian hereditary type. This compares favorably with the 35% figure reported by Hughson et al[4] and others.

Before trying to explain the pathology of hereditary deafness to Richard's parents, I wanted to refresh my own thinking on the anatomy and physiology of the inner ear. Although Stacy Guild in a recent personal communication said "We still do not know exactly how we hear," I was encouraged by the progress that has been reported in the past 15 years. At the 1937 meeting of this society, there was held under the direction of E. P. Fowler, Sr. a symposium on "The Neural Mechanism of Hearing." Participants in this symposium were Guild, Lorente, DeNo, Hinsey, Anson, Fletcher, Lewy, Lurie, Davis, Kobrak, Eyster, Hughson and Crowe,[5-6] a most distinguished group. Concerning the central connections of the eighth nerve, Hinsey pointed out that there is a dorsal and ventral nucleus probably of equal importance. He also pointed out that the medial geniculate body in addition to being a way station to the cerebral cortex is also an important center for auditory reflexes. This morphological observation was substantiated by the physiological studies on the cat as reported by Brogden et al.[7] They found that with one cochlea out there was a 4 db drop in hearing the same as with removal of one-half the cerebral cortex. By combining the removal of one-half the cortex and either the same or opposite cochlea there was an equal drop of about 15 db, showing that the crossed and uncrossed fibers were about of equal importance.

Culler et al[8] in 1943 confirmed their previous work on the frequency points in the guinea pig cochlea. Smith[9] also found essentially the same thing. Therefore, we might assume that in the human cochlea the higher pitches stimulate near the base with 3000 cycles about half way to the apex, 1000 cycles about 2/3 of the way up and 300 cycles about 4/5 of the way up.

Then it seemed in order to study the histological picture of reported specimens and try to correlate these findings with clinical observations, our anatomical knowledge and physiological experiments. Lurie[10] reported on the study of the temporal bones of congenitally deafened collies and dalmatians. He noted no abnormalities in the bone itself. The organ of Corti was degenerated, the scala media collapsed and the stria vascularis atrophied. There was some atrophy of the cochlear nerve and the spiral ganglia, collapse of the saccule with atrophy of the saccular nerve. In each instance, the utricle and semicircular canals seemed normal . In 1932, Fraser[11] classified cases reported until then and added one of his own. He pointed out that cases of complete absence of the labyrinth (Michel's type) are very rare. The next type is that with marked malformations of both the bony and membranous labyrinth (Mondini's type). His third classification were those cases in which the malformation is confined to the saccule and the membranous cochlea (Scheibe's type). He states that 70% of congenital cases are of this latter type. Here we are confused on account of terms. On the basis of caloric tests, all of the 59 true Mendelian hereditary cases in my series were of the sacculo-cochlear type.

Albrecht[12] reported on the histological findings in two cases but in only one of them was there a clinical history of deafness. He gives Otto Mayer credit for having been the first person to describe the pathological picture of the cochlea in a case of hereditary deafness. He makes no mention of the condition of the utricle or semicircular canals. His description of the cochlea is similar to that found in Lurie's dogs but emphasizes that there was more malformation near the apex than at the base.[2] In 1939, Albrecht[13] published the findings in a case of certain hereditary deafness in which he studied the cochlear nucleus (no mention as whether it was the dorsal or ventral one) and the vestibular apparatus as well as the cochlea. He found the semicircular canals normal and he could not ascribe changes in the nerve trunk or ganglion cells as more than normal variations.

Saxen[14] seems to be the first person to describe pathological changes in the eighth nerve and nuclear cells in his two cases. He concludes that they are secondary to the malformations in the cochlea. Wittmaack[15] calls the previous authors to task for assuming that changes noted in the scala media and the organ of Corti are the fundamental condition in hereditary deafness because he can produce

the same condition experimentally by the diffusion of guanadin solution through the round window.

Gauger[16] is the first author to approach this cochlea vs. cochlear nucleus problem on a sound basis. He used 16 normal hearing persons of varying age to establish a normal nuclear picture. Then he examined the nuclear region in six proven pathological cases and concluded that the nuclear region was normal in all of these cases. Altmann,[17] in a personal communication, describes the findings in two cases of probable inherited deafness, one of them without and one with changes in the osseous frame work of the cochlea. He is the first one to say that the changes were more marked in the basal than in the apical part of the cochlea.

In my series of 186 cases of bilateral congenital deafness, there was residual hearing in approximately twenty percent but this was invariably at the 500-cycle area or lower. Therefore, it is very hard for me to correlate this fact with Culler and Smith's cochlear points and the preponderance of the reported pathological pictures.

Having tried to organize our present knowledge of the pathology of hereditary deafness there still remains the answering of the last question asked by Richard's parents. "How can this be prevented in our next child?" Investigation of this problem led me down many blind alleys but there was one observation that seems worth reporting. Darlington[18] points out that not all hereditary conditions are transmitted in the chromosomes of the nuclei. Some are in the molecular structures of the cytoplasm and as such are subject to virus and chemical alteration. This has already been done in certain plants and certain pneumococci. He feels that ribo-nucleic acid can alter the structure of the cytoplasm and give permanent genetic changes in the offsprings. Desoxy-ribo-nucleic acid can similarily alter the nuclei.

He concludes his report by saying, "The frontiers that exist between the studies of heredity, development and virus infection are thus technical and arbitrary. New possibilities of analysis and experiment will arise when we have learned the password to take us across these barriers." Carrying this idea into the problem of hereditary deafness one wonders if chemical or virus alteration of either the male or female cell may not have accounted for some of the 68% in which there was no previous history. Also, it is not too much to

hope that chemical alteration of the cytoplasm of the male cell may eliminate such traits as we have been considering. There is much work to be done.

SUMMARY

1. The answer to the problem of hereditary deafness is still far from solution.

2. A clinical classification of congenital deafness is presented in order to avoid confusion in the use of certain terms.

3. The need for an increased number of uniformly processed specimens is very great.

4. Experimental study as to the possible alteration in the transmission of hereditary deafness may be of value.

10515 Carnegie Ave.

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XCV

THE NERVE-DEAF CHILD: SIGNIFICANCE OF RH, MATERNAL RUBELLA AND OTHER ETIOLOGIC FACTORS

VICTOR GOODHILL, M.D.
LOS ANGELES, CALIF.

The present study deals with an investigation of two recently isolated etiologic factors in infantile perceptive deafness. Thus, maternal rubella in the first trimester of pregnancy and fetal erythroblastosis resulting from Rh factor incompatibility are shown to be significant causes of infantile perceptive deafness. The method of investigation involved (a) an otologic study of approximately 77 cases of infantile perceptive deafness, (b) an analysis of 904 questionnaires addressed to the mothers of profoundly deafened children, and (c) an otologic study of 15 known erythroblastotic children.

The clinical material for this study was obtained from the Childrens Hospital, the John Tracy Clinic and from private practice. The questionnaires were obtained largely from parents in the correspondence course files of the John Tracy Clinic and also from the Mary E. Bennett School.

Previous Concepts Regarding the Etiology of Nerve Deafness in Children. Otologic literature at the turn of the century was rich in papers dealing with the etiology of congenital deafness, hereditary deafness and deaf-mutism. Inthe 1893 edition of Politzer's[26] text, a sizable bibliography and a comprehensive discussion of this subject is presented. Holger Mygind, Toynbee and Clarence Blake were credited by Politzer with numerous investigations on the subject. Studies detailing the numerous and complex anomalies of the cochlea, end organ, and auditory nerve found in deaf mute temporal

From the Department of Otolaryngology, University of Southern California School of Medicine; Childrens Hospital of Los Angeles; and the John Tracy Clinic.

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bones were presented by Alexander[1] Kerr Love, Hammerschlag and others. In addition to anatomic anomalies, scarlet fever and meningitis were considered important causes of deafness then as they are now. Typhoid, typhus, syphilis, cretinism and other diseases were given important status as causes. Today, such causes are obviously on the decline.

In spite of the well defined and studied cochlear aplasias, and the fine genetic studies of familial deafness, which together described only a small segment of the vast bulk of congenital deafness cases, etiology remained an enigma. True, most of these patients were classified in some way. Many were allocated diagnoses purely on parental suggestion. Thus the diagnosis of bronchitis, and pleurisy appear almost as often as measles, and pertussis on the records of such patients. Definitive knowledge of etiology has been fragmentary.

The present study dealing with transplacental cochlear invasion by the rubella virus and postnatal chemical destruction of auditory tracts by the icteric pigment of erythroblastosis may point the way to further modes of investigation into this problem.

Suggested Classification of Infantile Perceptive Deafness. The following classification has been devised largely for convenience and is based primarily upon current concepts of developmental defects. However, because of the theoretical knowledge regarding some of the etiologic factors, their exact position in the following classification must be considered tentative.

The somewhat ambiguous usage of the word congenital has prompted its omission from the classification.

I. Hereditary; Genetic (Gene Transmitted).
    A. Familial developmental anomalies of the auditory apparatus, both central and peripheral.
        1. Histologic aplasias
        2. Heredodegenerative

II. Acquired
    A. Prenatal
        1. Infections
            a. Spirochetal—Syphilis—transplacental
            b. Viral—rubella—transplacental
            c. Viral—others—transplacental

2. Acquired development anomalies. (circulatory —endocrine—metabolic).
3. Drug intoxications (quinine—salicylates)

B. Natal
1. Birth Injuries
2. Erythroblastosis fetalis—Rh factor—central (possible prenatal).

C. Postnatal
1. Meningitis
2. Acoustic neuritis—viral.
3. Encephalitis
4. Miscellaneous

SIGNIFICANCE OF THE RH FACTOR IN THIS PROBLEM

1. *History of Rh Factor Observations in Deafness.* In August, 1947, as a result of preliminary observations on a group of congenitally deaf children, the author[13] stated, "Rh factor disturbances may also play a part in the etiology of some of these cases. Several congenitally deaf children of Rh negative mothers are now being studied and will be reported more completely in the near future. Subclinical erythroblastosis in the newborn may have etiological significance in some cases of congenital perceptive deafness. It is quite possible that changes similar to kernicterus occur in the cochlea or in the ascending auditory tracts."

In a subsequent report before the American Academy of Ophthalmology and Otolaryngology (October, 1949) further material on this subject was presented with complete otologic and serologic details on 11 patients and their families. The theoretical background of the relationship between erythroblastotic kernicterus and the possible auditory pathway lesion was presented.

Since these two reports, the author has been encouraged by similar studies by Marcus[20] of the University of Illinois and Sataloff[29] of the University of Pennsylvania.

One of the sequelae of Rh factor incompatibility, namely, erythroblastosis with kernicterus may result in a bilateral perceptive deafness and other physical defects, the most common of which is athetoid spasticity. There is some evidence that deafness may occur without

spasticity. This etiologic factor is not a common one in deafness, but it nevertheless has great significance for two reasons. In the first place, it explains a certain small number of cases of perceptive deafness associated with spasticity which heretofore had no etiologic explanation. In the second place, it points to the possibility that other so-called unknown cases of deafness may be due to similar serologic phenomena as yet undiscovered.

2. *Otologic Examination of 15 Known Cases of Erythroblastosis.* This is a preliminary report of the study of a group of known recovered cases of erythroblastosis. From the files of Childrens Hospital, Los Angeles, records on 46 cases classified as erythroblastosis fetalis and admitted prior to 1946 were collected. This date was arbitrarily selected to exclude children under four years of age, to allow for possible audiometric study. An intensive search was made to locate these 46 cases, and subject them to otologic and audiometric study. Of these 46 cases, it was possible to locate and examine only 15 cases.

Otologic and audiometric examination of these 15 cases (known recovered erythroblastotics) yielded only two cases of significant perceptive deafness. Both of these children were spastics as well. Obviously such a small sampling prohibits any real numerical conclusions. The folowing two new case histories are therefore derived from this otologic survey of 15 cases of erythroblastosis. These cases have not been reported previously.

Case 1.—R. H. This child was first seen on April 5, 1950, at the age of eight years. He is the fourth sibling. The first and second children were perfectly normal. The third child died four days after birth, cause unknown. The patient, the fourth child, was born jaundiced, received several transfusions, and required hospitalization for two months. It was only recently determined that the mother is Rh negative, the father Rh positive and the child Rh positive.

Deafness and athetoid spasticity were noted shortly after birth.

Examination revealed no significant objective findings in the ears, nose or throat. There was evidence of athetoid spasticity. Audiometry revealed a bilateral perceptive deafness with an approximate bilateral threshold of 70 decibels in the speech frequencies (Fig. 1).

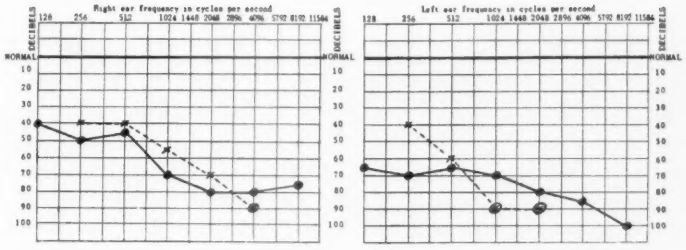

Right ear frequency in cycles per second
Left ear frequency in cycles per second
128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
DECIBELS
NORMAL
10 20 30 40 50 60 70 80 90 100

Fig. 1, Case 1(Rh Case).

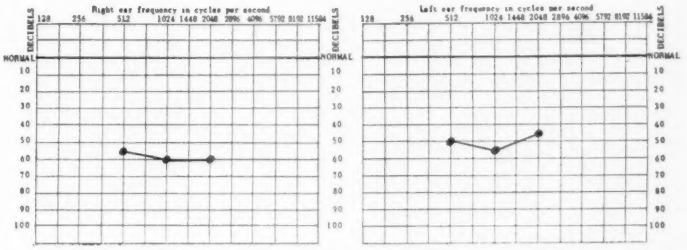

Right ear frequency in cycles per second
Left ear frequency in cycles per second
128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
DECIBELS
NORMAL
10 20 30 40 50 60 70 80 90 100

Fig. 2, Case 2 (Rh Case).

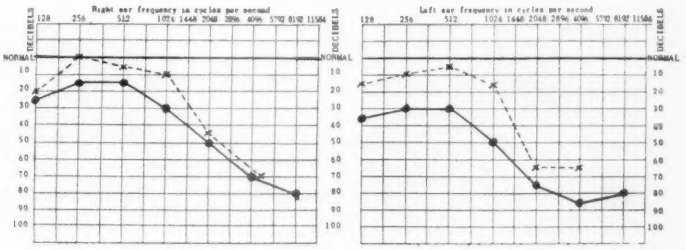

Right ear frequency in cycles per second
Left ear frequency in cycles per second
128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
DECIBELS
NORMAL
10 20 30 40 50 60 70 80 90 100

Fig. 3, Case 3 (Rh Case).

CASE 2.—T. S. This child was first seen on April 12, 1950, at the age of five years. He is the fifth sibling. The first four children are all perfectly normal and are twenty-one, eighteen, nine and seven years old respectively. There were two miscarriages between the second and third pregnancies, etiology unknown.

The patient was a full term delivery. Jaundice and high fever were noted on the third postnatal day. Spastic quadriplegia was noted shortly after birth. A diagnosis of kernicterus was made. The father and the child are Rh positive. The mother is Rh negative.

Examination revealed no signicant objective ear, nose or throat findings. Audiometric examination was difficult but revealed a fairly reliable 55 decibel bilateral perceptive defect in the speech frequency threshold (Fig. 2).

3. *Erythroblastosis and Perceptive Deafness without Spasticity.* In the previous report (1949) three such cases were described. The following is an additional case recently studied.

CASE 3—C. E., a five year old white female was first examined on December 23, 1949, with the history of retarded speech and poor hearing which had been noted since infancy. There had been a recent upper respiratory infection with noticeable deterioration of the previous hearing level.

Examination revealed moderate bilateral serous otitis media with chronic hypertrophic tonsillitis and adenoiditis. An audiogram on that day revealed a bilateral mixed type of deafness with a symmetrical bilateral high tone perceptive defect. There was no other significant history as far as this child is concerned, with the exception of the following specific Rh history.

The mother is a known Rh negative and the father is a known Rh positive. Their first child was born in April, 1940, perfectly normal in every way. During the summer of 1941 there was a miscarriage at six weeks gestation necessitating a transfusion to the mother. On August 15, 1942, a second child was born by an emergency section following a placenta previa. On December 9, 1944, the present patient was born by caeserean section, three weeks premature. The child was born markedly jaundiced, and received one transfusion in the hospital. There were no evidences of spasticity, either in the patient or in her sisters. The sisters have no difficulty in hearing.

SPECIAL QUESTIONNAIRE REGARDING THE CAUSES OF DEAFNESS

NAME OF MOTHER..... Age.....

ADDRESS.....

CHILD'S (Patient's) NAME ..... Age.....

1. Was child born deaf?.....
2. Did child seem to hear in the first year of life and then lose hearing later?.....
3. Did deafness seem to follow any specific illness of child?.....

   If so, what illness?..... At what age?.....
4. Does your child seem to have noises in ears?.....
5. Does your child ever get dizzy and lose balance?.....
6. Does your child seem to have hearing for certain tones?.....
7. Does your child respond to loud vibrations?.....
8. Did you take any quinine, or any other unusual drug, during pregnancy?.....
9. How many other children have you?.....
10. Are any of your other children deaf?.....
11. Did you or your husband have any deafness in your families?.....
12. Are you and your husband related?.....

Fig. 4.

Serologic study of the parents was repeated and showed very definitely that the father was Rh positive and the mother Rh negative. The Coombs test for indirect antibody titer revealed a strongly positive reaction in the mother on February 17, 1950.

On January 13, 1950, a tonsillectomy and adenoidectomy was performed. On January 26, 1950, the nose, throat and ears were essentially normal. There was no evidence of further serous otitis media. An audiogram done on that day revealed a definite improvement in some of the lower frequencies. However, the high tone perceptive impairment persisted as shown on the audiogram. Because of the very definite erythroblastotic history in this child, and the inability to attribute the deafness to any other known etiologic factors, it may be assumed that the deafness in this child is due to erythroblastotic subclinical kernicterus.

4. *Questionnaire Analysis of the Incidence of Erythroblastosis.* Erythroblastosis fetalis is presumed to occur in approximately one out of 250 births, according to Diamond,[7] Potter[27] and others.

SECTION "A"

1. What is your Rh blood type?.....
2. What is your husband's Rh blood type?.....
3. How many miscarriages did you have?.....
4. Was your child anemic (pale) or yellow jaundiced at birth or shortly after birth?.....
5. Did your child ever have convulsions or a spastic condition?.....
6. Did you personally have anemia during or following this pregnancy?.....
7. Was a blood transfusion necessary for either you or your child?.....
8. Were any of your other children anemic or yellow jaundiced at birth, or did they require transfusions?.....

SECTION "B"

1. Did you have German measles or any rash during your pregnancy?.....
   Give details regarding type of rash and exact month of pregnancy?.....
2. Did you have any other illness during pregnancy—such as mumps, influenza, toxemia, etc.?.....
3. Does your child have any other condition in addition to deafness—such as cataract, or other bony, muscular, or eye difficulties?.....
4. Did your child (the patient) have any kind of measles?.....
5. Did any of your other children have any abnormalities—physical, mental, eye, or otherwise?.....

Fig. 5.

In the present questionnaire answered by 904 mothers of profoundly deafened children, it was deemed reasonable to consider these 904 children as a fairly random sample of the population. Included in this sampling were children deafened by maternal rubella, meningitis, measles, scarlet fever, familial hereditary traits and many other known and unknown causes. The only common denominator for this group of 904 children was deafness.

A series of questions designed to elicit the possible diagnosis of erythroblastosis was included in this questionnaire. The answers to these questions were charted, analyzed and evaluated as to the possibilities of the affected child being an erythroblastotic (Figs. 4 and 5).

This study was replete with problems. Inasmuch as the questionnaire was answered in early 1948, knowledge of the parental Rh serology was grossly lacking, the majority of these children being born in 1942, 1943 and 1944. During these years, only a scant

knowledge of the Rh factor was possessed by the medical profession. Consequently direct serologic answers were infrequently found in the questionnaire. Attention was directed primarily to other answers which would reflect the possibility of erythroblastosis. Such factors as concurrent spasticity, postnatal jaundice and anemia, sibling spasticity, etc. were used in the evaluation.

Obviously, any attempt to determine a diagnosis of erythroblastosis or kernicterus from questionnaire answers must be considered presumptive. The following findings, therefore, are offered only in that light. They should not be considered absolute observations. However, it is apparent that even in such an analysis, certain striking correlations are evident.

The following criteria were set up for this evaluation. Eight important circumstantial facts elicited in the answers were listed as follows:

1. Known Rh negative mother, and Rh positive father.
2. Patient jaundiced at birth
3. Patient transfused at birth
4. Patient is spastic
5. History of multiple miscarriages
6. Sibling postnatal jaundice
7. Sibling spasticity
8. Sibling mortality

Whereaver six out of eight answers were positive, the chart was labeled "probable erythroblastotic." Wherever four or five out of eight were positive the chart was labeled "possible erythroblastotic." Less than four out of eight were merely included in a "doubtful" category.

It is, of course, very likely that some cases were not included because of the lack of knowledge of Rh serology on the part of the parents. Subsequent investigations may reveal larger figures because of improved prenatal knowledge of Rh serology.

This analysis yielded the following data. Of the 904 questionnaires studied there were 27 probable, 4 possible and 9 doubtful cases. Of the 27 probable cases, 9 were spastics.

In the opinions of several serologists, the criteria established for "probable erythroblastotic" were considered realistic and plausible.

According to the present conservative figure of erythroblastosis occurring approximately once in 250 births, one would not expect to find more than four cases in 904 children. In view of the mortality in erythroblastosis, even 4 cases is a high estimate in 904 living children. It is significant, therefore, to find 27 probable, 4 possible and 9 doubtful cases in this random sampling of 904 children who have only one thing in common, namely deafness.

5. *Pathological Studies of Localization of this Lesion.* At the present time, the localization of this lesion remains in doubt although the author has predicated that the lesion occurs in the cranial nuclei, thus describing the deafness resulting from kernicterus as nuclear deafness. Several brains are now being studied by Dublin,[8] and several temporal bones are being studied by Wolff,[35] with a view towards the possible solution of this problem of localization. This pathological study is a long range problem and will require years for definitive information.

6. *Place of the Rh Factor in Infantile Nerve Deafness.* The evidence listed above establishes Rh factor incompatibility as one cause of infantile perceptive deafness. The mechanism probably involves the toxic effect of ieteric pigments (bilirubin, etc.) upon the cranial nuclear masses traversed by the auditory radiations, as a sequel of erythroblastotic kernicterus. The central localization corresponds to kernicteric extrapyramidal tract involvement which produces athetoid spasticity.

The Rh factor thus takes its place in the etiologic classification of infantile perceptive deafness. This position is not a major one. It does not approach maternal rubella, meningitis, and the exanthemata in numerical importance. It is, nevertheless, a significant cause and one which may point to other serologic factors in the etiology of infantile deafness.

SIGNIFICANCE OF RUBELLA IN INFANTILE PERCEPTIVE DEAFNESS

1. *History of Australian Rubella Observations.* In 1940 a severe and widespread epidemic of rubella (german measles) occurred in Australia. The epidemic apparently started in 1939 and ended in

January 1941. Its height was during July and August of 1940. In 1941, Gregg[14] reported a number of cases of cataract in the children of mothers who had rubella during the first trimester of pregnancy. An investigation of this maternal rubella history revealed that most of these women had very severe german measles with adenopathy, sore throat, involvement of ankle and wrist joints and general constitutional symptoms. The rash was pleomorphic, macular, scarlatiniform and toxic-erythematous.

The initial report of complications in the children was confined to the discovery of cataracts. Within a very short time, however, Gregg's paper was followed by several other papers from Australia in which congenital cardiac lesions and deafness were reported as further sequelae. Notable among these were the papers of Swan, Tostevin and Black,[31] Carruthers,[4] Welch,[33] Martin,[21] Winterbotham,[34] Evans,[10] Murray,[23] Mann,[19] Scholes, and B. Hiller[17]

In 1946, Gregg[15] reported an incidence of 111 cases of deafness in 130 cases of rubella. Of this same group of 130 cases, there were only 38 with heart disease and 23 with eye lesions.

The Australian investigators must be credited with the brilliant analysis of an unusual clinicopathologic association which turned the attention of the entire world to this previously unknown factor.

In 1946, Winterbotham[34] who studied the cardiac defects associated with maternal rubella, noted that epidemics of rubella are known to occur every third or fourth year in Queensland. It is a remarkable fact that practically every fifth year for the past 42 years there has been a wave of increase in the number of deaf children occurring in that community.

A surprisingly high incidence of congenital dental abnormalities was reported by Evans[10] in 1947. In 30 out of 67 children whose mothers had maternal rubella, major congenital dental abnormality was demonstrated. Shark-like and pointed incisors accompanied by enamel hyperplasia was the most frequent group of dental anomalies.

In 1946, a very detailed analysis of the sequelae appeared by Swan, Tostevin and Black.[31] Abnormalities found included cataracts, deaf-mutism, heart disease, microcephaly umbilical hernia, bifid uvula, mental deficiency, epilepsy, speech defects and concomitant

strabismus. They theorized on the possibility that the virus may gain access to the embryo earlier, that is during the relatively long incubation period (approximately 18 days) of the disease, thus making it possible that the attack on the embryo may be before the rash. They also mentioned that there is suggestive evidence that infection of the mother with rubella prior to fertilization of the ovum may sometimes be followed by congenital defects.

Gregg[14] recorded a case of congenital cataract in a baby whose mother had contracted rubella three months before pregnancy. He also reported later, a case of deaf-mutism in which the infection had presumably preceded conception. While there are questions regarding the authenticity of these occurrences, nevertheless, they raise interesting theoretical considerations.

The most recent survey of the deafness problem following maternal rubella in Australia was made by Murray[23] in 1949. This was a very thorough audiologic study of 105 children considered to be nerve-deaf due to maternal rubella. He found that there were two peaks of the occurrence of maternal rubella deafness; one was at six weeks gestation and one at three months, and suggested that this corresponded with the fact that the main development of the cochlea begins at about six weeks, while the three month period corresponds closely with the rapid development of the organ of Corti. He could find no difference in the degree of deafness occuring when the rubella was contracted at six weeks and at three months. The general pattern of deafness observed was one in which the loss for low tones was greater than the loss for high tones. He laudably deplored the term deafmute in this survey. It was his opinion that most of these children are only moderately to severely deaf but not totally deaf and that most of them can benefit very well from the use of hearing aids. He found the average hearing loss over the speech range in all ears at about 72 decibels and for the better ears, 65 decibels.

Another fine recent report was from Tasmania by Hiller[17] in 1949, who studied 42 congenitally deaf children of which 32 were presumed to be of rubella origin. His audiometric findings were similar to those of Murray.[23]

2. *History of British Rubella Observations.* In 1946, Martin[21] reported a study of 102 deaf children born in 1940 and 1941. In

36 of these cases a history of rubella in the mother during the first four months of pregnancy was obtained. Details regarding ocular and cardiac lesions were not given but they were mentioned as being present. Apparently there was an epidemic of rubella in Britain during 1940 and 1941.

In 1947, Clayton-Jones[6] reported an extensive study from England detailing the findings in a number of deafened children, all of whom were born during the severe English rubella epidemic of 1940.

In a critical analysis of maternal rubella as a cause of defects, Parsons,[25] in 1946, raised several pertinent questions regarding the authenticity of the association. He wonders why measles (rubeola) does not produce such defects but concludes that in measles "there is always the possibility that the effect of the more severe virus infection was to produce abortion."

3. *History of American Observations.* In 1944, Reese[28] reported three cases of congenital cataract and congenital heart disease occurring in mothers who had contracted rubella in the first months of pregnancy.

In 1944, Erickson[9] of Pasadena reported 11 cases of congenital ocular malformations, nine of which also had congenital cardiac defects.

The original reports of maternal rubella as a cause of congenital defects emphasized the predominance of ocular and congenital cardiac defects, and largely ignored the much higher incidence of deafness. The reason was understandable inasmuch as these reports were usually made when the children were less than two years of age. In the majority of these instances, deafness had not yet been diagnosed and the presenting findings were cataracts and the congenital cardiac states. The later Australian statistics as well as those of Britain and the United States showed clearly that deafness was by far the most common sequel of maternal rubella, and that congenital cataract and congenital cardiac disease were relatively infrequent sequelae.

In 1945, Altmann and Dingman[3] reported a case of congenital deafness and cataracts following rubella in the mother.

In 1949, Hopkins[18] gave an excellent statistical study of 5 cases of deafness with a history of maternal rubella. Her study in-

dicated the presence of certain developmental arrests in the rubella deafened group as contrasted with a group of controls.

Ober, Horton, and Feemster[24] in a study of 54 pregnancies occurring in adult females who had rubella in 1943 in Massachusetts found that infants with abnormalities were observed in eight, abortions occurred in nine. Eleven of the 17 abnormal pregnancies were in women who contracted rubella during the first trimester. Furthermore, it was found that of nine pregnancies reported by physicians in response to questionnaires, only three produced normal infants and eight of the nine women contracted rubella in the first trimester.

A similar study by Aycock and Ingalls[2] was very pointed in its reference to rubella as a cause of potentially serious diseases still unrecognized and also speculated regarding the role of poliomyelitis and other virus diseases as etiologic agents of many fetal diseases and developmental anomalies.

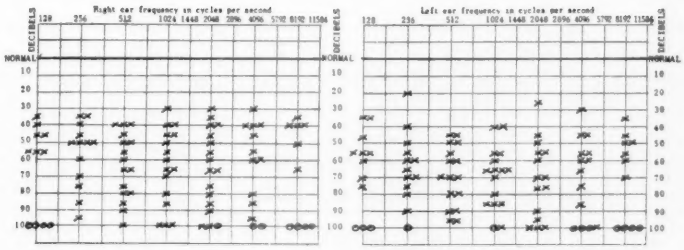
In 1947, Fowler[11] stressed the importance of maternal rubella in congenital deafness.

In 1948, MacFarlan[22] in writing on the congenitally deaf child drew attention to the severity of this problem by stating "recent investigations show that rubella in the pregnant mother is a frequent cause, even though the attack may be mild."

In several recent American texts on otolaryngology, maternal rubella is given definite mention as a cause of deafness.

4. *Pathological and Embryological Considerations.* The auditory placode is first visible in the two week embryo. By the sixth week there is a saccule and a coiled cochlea.

According to Guggenheim[16] at two and one-half months there is a thickening on one wall of the ductus cochlearis. By the third month this thickening has become two thickenings, an inner larger one which will form the limbus spiralis, inner sulcus, tectorial membrane, inner cell of Corti and inner pillar of Corti. The outer smaller thickening will form the outer pillar, the outer cells of Corti, etc. At this three months period there is nothing to suggest the definitive Corti organ except for a small space between the two thickenings, which becomes the tunnel of Corti. From the fourth fetal month on the development is rapid, and the organ of Corti is completed by the sixth month at which time the fetus actually hears.



SCATTERGRAM OF AUDIOGRAMS OF 16 PATIENTS WITH PERCEPTIVE DEAFNESS WITH MATERNAL RUBELLA HISTORY.

Fig. 6.

It is easy to see, therefore, that transplacental virus infections occurring during the first three months of pregnancy may easily influence the developmental state of the human cochlea and that the extent of involvement will depend upon the stage of development as well as upon the intensity of the infection. The vestibular apparatus is apparently uninvolved by the virus of rubella. The cochlea is a more highly differentiated organ, which may well explain its greater susceptibility to noxious stimuli over that of the vestibular apparatus. The phylogenetic status of the vestibular apparatus is millions of years older than the cochlear apparatus. Organs of recent phylogenitic development are not as resistant as the older organs.

5. *Present Rubella Study.* The following findings on rubella were isolated by (1) a detailed otologic study of 19 personal cases and (2) the analysis of 186 cases obtained from the questionnaire sent to 904 mothers of profoundly deafened children.

Of a series of 77 consecutive cases of bilateral perceptive deafness in children, 19 cases gave a history of maternal rubella.

Nineteen cases were examined in detail otologically. The accompaning scattergrams illustrate the distribution of the audiometric curve in these cases.*

*Lack of space prevents the reproduction of the nineteen individual audiograms which accompanied the mms. [Ed.]

No significant conductive impairments were found. The audiometric pattern was almost always a purely perceptive loss. As can be seen from the scattergram (Fig. 6) there is a rather widespread distribution of the threshold levels. The majority of the curves are in the 40-70 decibel range. Recognized ocular defects were present in two of the 19 cases. Recognized cardiac defects were present in 3 of the 19 cases. Of these 19 children, 1 was born in 1936, 5 in 1943, 5 in 1944, 5 in 1945, and 3 in 1946.

6. *Analysis of Rubella Responses in Questionnaire.* A. Incidence of Rubella as a Cause of Deafness.

In December 1947, questionnaires (Fig. 4 and 5) were sent to the mothers of some 1,500 profoundly deafened children. The names had been obtained from the files of the correspondence course division of the John Tracy Clinic. The geographic distribution of the group was widespread, including almost every state in the country and several foreign countries, California, however, predominated.

During the first four months of 1948, 904 legibly and intelligently answered questionnaires were received. This material has been subjected to various types of analysis.

In 186 of 904 cases, positive responses to the rubella group of questions were sufficiently clear and definite to warrant their inclusion in the group of "maternal rubella etiology." A few cases in which other potential etiologic factors were present were not included. Thus, several of the children with a history of maternal rubella who also had meningitis in the first and second year of life were excluded from this analysis.

Thus, 20.5% of this entire series of 904 cases including infantile perceptive deafness of all types, may be attributed etiologically to maternal rubella. This percentage must be tempered, however, with the knowledge that the sampling may possibly contain a weighted number of rubella cases because of the war epidemic of rubella in this country.

Age Distribution and Epidemiological Correlation (with the collaboration of D. E. Wheeler, Ph.D. and John M. Chapman, M.D.) The age distribution on the basis of reported ages was studied in the total deaf group and rubella deaf group. The numerical values appear in Fig. 7 and are represented graphically in Fig. 8.

AGE DISTRIBUTION OF TOTAL AND RUBELLA DEAF GROUPS (AGES 1-10)

AGE	TOTAL DEAF	RUBELLA DEAF
1	18	
2	57	4
3	122	18
4	280	101
5	154	26
6	92	18
7	54	9
8	42	1
9	20	
10	10	
TOTAL	849	177

Fig. 7.

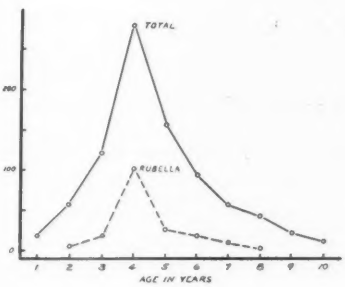


Fig. 8.

The mean age for the total group is 4.63 years. The mean age for the rubella group is 4.38 years. The standard error of the mean based on 849 cases is 0.06 years. It was proposed to statistically test the hypothesis that the mean age of the rubella group differed significantly from the mean age of the total group.

For this purpose, and on the assumption that the distribution does not seriously depart from normality a "t" test was applied. A ratio of 4.1 was obtained; this ratio means that one may accept (at the 1% level of confidence) the hypothesis that the mean age of the rubella group is significantly different from the total age group.

This difference, however, while statistically significant is not great. It amounts to only 0.25 year or 3 months difference in mean ages. Simple comparison of the distribution curves for the total deaf group and the rubella deaf group shows a striking similarity. Peaks for each curve center around the age of four years.

Inasmuch as the epidemiological connection between rubella and its sequelae has been studied elsewhere, it was decided to compare this age breakdown with known data on rubella epidemics in Los Angeles.

Fig. 9 shows a 25 year study of the epidemiology of rubella in reported cases per 100,000 population. It will be noted that sharp peaks occurred in 1928, 1935, and 1943.

Inasmuch as the questionnaire was completed during the first months of 1948, it is reasonable to assume that a child reported as age four was born in early 1944. Therefore, the time of conception was probably the spring of 1943.

It will be observed from Figs. 9 and 10 that the greatest epidemic peak occurred in March, April and May of 1943. It is, therefore, not unlikely that the four year old peak in the rubella deaf group is correlated with the known epidemic peak of rubella at the time of conception of this 4 year old group.

Of still greater interest, however, is speculation concerning the similarity in 4 year peaks between the rubella deaf group and the total deaf group. The total deaf group includes approximately 20% rubella deaf and 10% meningitis deaf. The remaining 70% include

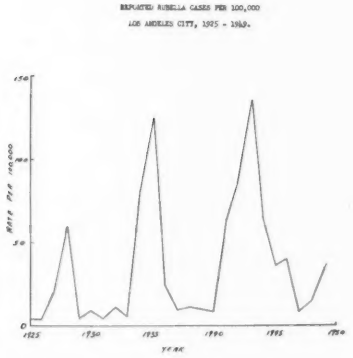

REPORTED RUBELLA CASES PER 100,000
LOS ANGELES CITY, 1925 - 1949.
RATE PER 100,000
150
100
50
0
1925
1930
1935
1940
1945
1950
YEAR

Fig. 9.

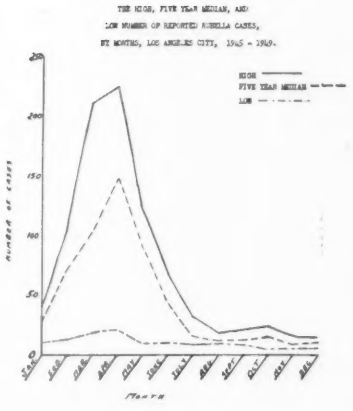

THE HIGH, FIVE YEAR MEDIAN, AND
LOW NUMBER OF REPORTED RUBELLA CASES,
BY MONTHS, LOS ANGELES CITY, 1945 - 1949.
HIGH
FIVE YEAR MEDIAN
LOW
NUMBER OF CASES
200
150
100
50
0
MONTH

Fig. 10.

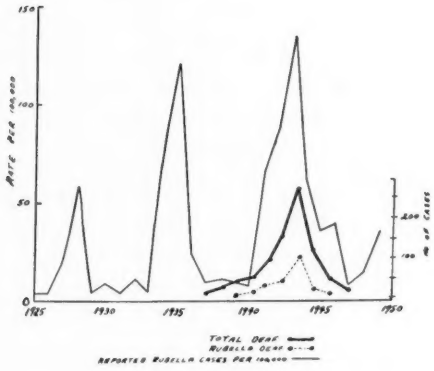


Fig. 11.

a vast array of known and unknown etiologic factors. Is it not possible that in this 70% there exist a significant number of undiagnosed rubella deaf, as sequelae of the same 1943 epidemic? (Fig. 11).

It is interesting also to note that our epidemic in 1943 followed the Austrialian and British epidemics of 1939, 1940 and 1941.

Month of Gestation. Information regarding the month of gestation was available in 177 of the 186 rubella cases. The distribution is charted below (Fig. 12).

NUMBER	MONTH OF GESTATION
31	1ST
82	2ND
50	3RD
10	4TH
2	5TH
2	6TH

Fig. 12.

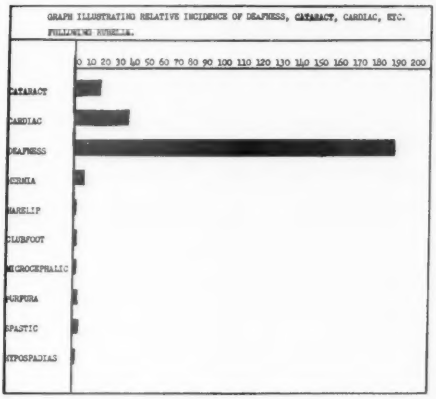


Fig. 13.

In 82 out of 177 or 46.3%, rubella occurred in the 2nd month of gestation. In 31 out of 177 or 17.5% rubella occurred in the first month of gestation. In 50 out of 177 or 28.2% rubella occurred in the third month of gestation. Thus, the second month appears to predominate in the distribution of cases.

Incidence of Defects other than Deafness. Of the 186 deaf children with a history of maternal rubella, 33 or 17.7% had congenital heart disease and 13 or 6.9% had congenital cataract. Other defects occurred sporadically as shown in the following table (Fig. 13).

Deafness	186
Congenital cardiac	33
Cataract	13
Hernia	4
Harelip	1
Clubfoot	1
Microcephalic	1
Purpura	2
Spastic	3
Hypospadias	1

Conclusions of Rubella Analysis. 1. Rubella (german measles) occurring in a mother during the first three months of pregnancy may cause serious sequelae in the child. The most serious and most common sequel is a bilateral perceptive deafness of sufficient severity to produce a major hearing disability. Less common sequelae are congenital cardiac defects, congenital cataracts, congenital skeletal and other defects.

2. The occurrence of maternal rubella during epidemics will naturally produce great variations in the frequency with which rubella will be found to be a cause of infantile perceptive deafness. It may be assumed as a result of the 1942-1943 epidemic that the incidence of rubella as a cause of all cases of infantile perceptive deafness during this 1948 analysis accounted for 20.5% of cases. This is by far the largest percentage of cases of any one disease heretofore considered as a cause of congenital perceptive deafness. What this percentage will be in future compilations in the next few years remains to be seen. However, it undoubtedly will still be high, much higher than has heretofore been realized.

Preventive Medical and Public Health Considerations. Rubella has traditionally been considered the least important of the common exanthemata by physicians and epidemiologists. The growing statistics on maternal rubella as a serious cause of disabling and crippling defects in the newborn must change this benign attitude to rubella.

As mentioned before, earlier reports on maternal rubella stressed congenital cataract and cardiac diseases and largely ignored deafness. The demonstration that deafness is a far more common sequel than either cataract or heart disease should provoke greater interest in the public health aspects of this disease.

Three important questions await solution. (1) Should all unmarried girls be intentionally exposed to rubella to insure an active immunity to the disease? (2) Should non-immune pregnant women exposed to rubella in the first few months of pregnancy be given passive immunity by the use of convalescent or hyperimmune sera? (3) Should pregnant women who develop rubella in the first few months of pregnancy be aborted therapeutically?

Significance of Meningitis in This Survey. In previous studies of large numbers of deafened children, meningitis has always occu-

pied a prominent position on the roster of etiologic factors. In an attempt to evaluate its significance in the present study, the answers relating to known causes were studied.

Of the 904 questionnaires, positive answers for meningitis as a cause were given in 93 instances. These 93 cases of meningitis were described as follows:

Meningitis (epidemic—meningococcic—unclassified)	61
Meningitis (pneumococcic)	19
Meningitis (influenzal)	12
Meningitis (tuberculous)	1
Total Meningitis Cases	93

Meningitis, as a group, therefore was present in 10.3% of the total case material.

It is interesting to compare this figure of 10.3% for meningitis with the 20.5% incidence of maternal rubella as a potential cause.

Significance of Other Etiologic Factors in This Survey. The significance of hereditary (genetic) deafness, the exanthemata, quinine and salicylate intoxication, and many other factors were subjected to study in this questionnaire. However, definitive conclusions are impossible at the present time. The reliability of parental observations in regard to these factors is open to great question. Consequently, no conclusions can now be drawn from this material.

CONCLUSIONS

Two groups of patients with infantile perceptive deafness were studied with reference to etiologic factors. One group was examined otologically and audiometrically. The other group, much larger, was analyzed by means of answers to a questionnaire.

Maternal rubella in the first three months of pregnancy was the potential cause of deafness in 186 (20.5%) of the 904 cases studied in the questionnaire.

Inasmuch as this study involved a large number of children who were conceived during a known rubella epidemic in this country, the above figures may represent a temporarily high level and not an average or mean value over a larger period of years.

The striking role of maternal rubella as an apparent cause of deafness evokes important public health recognition of this supposedly benign disease. The questions of intentional exposure of unmarried girls, use of preventive and therapeutic sera, and therapeutic abortion merit the serious consideration of the entire medical profession.

Further evidence has been produced to add erythroblastosis (due to Rh factor incompatibility) to the list of etiologic factors responsible for infantile nerve deafness.

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XCVI

THE OTOLOGICAL CONCEPT OF BELL'S PALSY AND ITS TREATMENT

JOSEPH A. SULLIVAN, M.B.

AND

J. BRYDON SMITH, M.D.

TORONTO, CANADA

It is said that seventy-five percent of cases of Bell's palsy recover spontaneously, and therein, we think, lies the cause of life long disfigurement for many people.

The twenty-five percent of cases who do not recover are forced to go through life with this disfigurement that only those who recover can understand. We should regard Bell's palsy rather from the point of view of those who do not recover spontaneously, and say that in twenty-five percent of cases the present usual line of treatment results in complete failure and the patient is permanently disfigured.[1] This is a more arresting point of view and it is likely to result in a considerable diminution of such failures; that the treatment of any surgical condition should have so high a failure rate as twenty-five percent should of itself call for attention and inquiry. In every case of Bell's palsy the safe procedure is to presume that it will not recover, unless something more than an attitude of masterly expectancy is adopted.

Progress is not always made by the presentation of new ideas or discoveries, but depends much more on newer comprehensions of established facts, and particularly on the co-ordination and interpretation of old ideas.

The name, Bell's palsy, has been applied indiscriminately to infranuclear lesions of the nerve causing facial paralysis. Sir Charles Bell[2] in a paper read before the Royal Society in 1821, stated, "That in

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Read before the American Otological Society, San Francisco, California, May, 1950.

animals where the facial nerve was severed they suffered no pain, but on exertion the side of the face on which the nerve was divided did not move as did the other side." This led Bell to call the seventh nerve the nerve of respiration of the face. He was critisized for this, but maintained his view by pointing out that muscles of the chest and shoulder were used when respiration became embarrassed. Bell established the concept of the motor seventh and sensory fifth which is today accepted as a matter of course. The condition as we now know it and which we call, Bell's palsy, is not that originally described by Sir Charles Bell. We like to look upon it as that condition which occurs in an apparently normal and healthy individual, and coming on spontaneously in which he finds he has lost the power of movement of one side of his face. We should be careful to differentiate the cases which are of a zoster form of infection and are supposed to involve the geniculate ganglion from the definite group under discussion in this paper, namely, a lesion involving the nerve within, at, and just without, the stylomastoid foramen. If there were any facial cutaneous fibres in man, we should find in every case of Bell's palsy as complete a cutaneous anesthesia of its alleged auricular zone as any physician ever found on the trigeminus area after a complete Gasserianectomy.[3]

Gowers[4] regarded Bell's palsy as a definite disease group and said, "The features of these cases are so uniform, allowance being made for differences in degree, that we are justified in regarding the pathologic condition as the same in all, a neuritis within the Fallopian canal."

It is a brutal knock-down blow by an acute gross lesion which irritates, compresses, or strangles every structure which it meets, whether it be nervous, vascular, or lymphatic. It has been previously held that the causative factor of this lesion was due to cold and often erroneously termed, rheumatic. Yet at the same time the majority of cases of facial paralysis seem to be of this nature, although, often no special exposure to cold or such can be traced. There is a seasonal incidence more common in the winter than in the summer, more common in the male than the female, a tendency to a familial history, and recurrences. The onset of the paralysis is usually rapid, and reaches its height in a few hours. The duration varies according to the degree and character of the nerve lesion, which may be severe or slight.

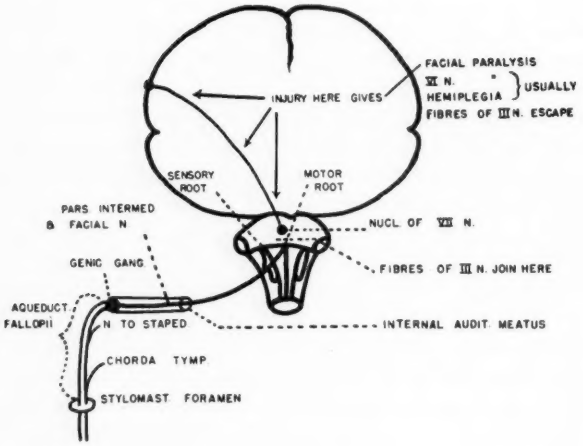

Fig. 1.—Diagrammatic representation of the course of the facial nerve—after Morris.

Anatomy. It is essential that we trace the course of the facial nerve fibres from the cortex, to their distribution to the muscles of the face in order to be able to localize with any certainty the position of a lesion causing facial paralysis[5] (Fig. 1). It must be kept in mind, however, that it is only surgically possible to combat lesions of this structure which occur in the Fallopian canal. Starting from the cortex the motor fibres run down to the seventh nucleus, situated deep in the lower portion of the pons, and they emerge as the motor root at the lower border of the pons, between the olive and the restiform body. Just before the motor root leaves the pons it is joined by fibres from the third nucleus, which supply the following muscles; the orbicularis palpebrarum, corrugator supercilii, and the anterior belly of the occipitofrontalis. Lesions occurring, therefore, in the cortex, or in the course of the fibres to the nucleus or a little beyond will cause paralysis of the muscles of the face with the exception of these three; a questionable deafness due to paralysis of the stapedius muscle, and no loss of taste in the anterior two-thirds of the tongue. There will be no dryness of the mouth, as the fibre supply of vasodilator nerve impulses to the submaxillary and sublingual glands are not involved. The electrical reactions will be normal. The lesion

may be traumatic, embolic, or due to new growth, and usually entails paralysis of the sixth nerve and hemiplegia. The diagnosis of a lesion in this area is by the fact of the escape of the three muscles mentioned.

Proceeding further along the course of the motor root the next part of importance is the internal auditory meatus. Occasionally the nerve is injured in the removal of an acoustic tumor. The injury in this portion will give a complete facial paralysis and deafness, apart from the deafness caused by the acoustic tumor. Conversely the sensory root which is closer to the eighth nerve than the motor root is, may be injured. In the internal meatus, that is up to the geniculate ganglion, injury is most commonly due to fracture of the base of the skull, and nerves sixth, seventh, and eighth, are usually involved.

The next important diagnostic site from the surgical point of view, is from the geniculate ganglion to the stylomastoid foramen, which is the Fallopian aqueduct. In the lower portion, that is the vertical segment, which is between half and three-quarters of an inch long the nerve runs through dense bone, and it is in this portion only, that Bell's palsy occurs. In my opinion, the chorda tympani nerve is the only important anatomical branching from the facial nerve that allows us to accurately localize the site of the lesion, and this is done by testing for the sense of taste on the anterior two-thirds of the tongue. Lesions of the nerve after it leaves the stylomastoid foramen are usually traumatic during operation or accident, or due to new growth of the parotid gland and are easily distinguishable.

The blood supply to the facial nerve in its Fallopian canal is derived from the following sources: The stylomastoid artery enters the stylomastoid foramen in relation to the facial nerve and supplies the tympanic cavity, mastoid cells and semicircular canals. It is carried onward in the Fallopian canal to anastomose with the petrosal branch of the middle meningeal artery. This artery runs practically throughout the whole length of the canal and supplies the neurilemma of the facial nerve. A branch of the middle meningeal artery called the petrosal artery enters the facial canal supplies the facial nerve and anastomoses with the stylomastoid branch of the posterior auricular artery.

It is the fundamental purpose of this presentation to clarify, if possible, the underlying etiological factor for the production of

this lesion. From the following table of my series of cases it is seen that over a period of twenty years, operative interference has been carried out in a hundred and twenty-five cases of Bell's palsy.

TABLE 1.—VARYING TYPES OF FACIAL NERVE PARALYSIS (SULLIVAN 1930-1950)

	CAUSES	CASES
1.	Bell's Palsy	125
2.	Mastoid Operations	515
3.	Auricular Herpes Zoster	17
4.	Mastoid Disease:	
	(a) Acute Otitis Media	30
	(b) Chronic Suppurative Otitis Media	58
5.	Late Post Operative	24
6.	New Growth	14
7.	Skull Fracture	22
8.	Others:	
	(Hypertensive Vascular Disease)	5

From this experience, gained by actual observation of the nerve under a dissecting microscope on the operating table, the following deductions are made:

Bell's palsy is an edematous neuropathy of the distal segment of the facial nerve, a dysregulation of the circulation.[6] What sense is there, for instance, in speculating on how essential are drugs or vitamins to the young fibre sprouts, if one does not even know to what extent the latter depend on local sources for their oxygen and nutrient supply. The capacity of a nerve to regenerate is subject to constitutional biological limitations.[7] Neither the biological process of nerve regeneration nor the various techniques used to facilitate it for the sake of functional nerve restoration are indivisible wholes. All components of a nerve and of its surroundings, axons, sheath cells, fibroblasts, macrophages, etc., all these contribute in some degree to the outcome of nerve regeneration, and their various peculiarities and requirements want to be taken into account. The functional restoration of a nerve is a composite product of many and diverse interlocking phenomenon and no one of them can be singled

out as the master key to the control of the others. It has become fairly plain that there can be no physical or chemical agent, no drug that would be universally beneficial to all component processes of nerve regeneration alike. In the light of these realities as we now know them, many successful and empirical procedures of the past find a splendid ex post facto justification; any method of nerve repair will, therefore, have to be examined and all its consequences rated according to the balance of the good and harm it does. Methods of nerve repair must aim not at stimulating nerve growth, but providing it with the conditions under which it can best realize its own potentialities. In last analysis, it is the nerve that has to do the repairing and it seems that the only way in which nerve regeneration can be effectively promoted, is to avoid the natural hazards that tend to depress it. These handicaps operate on a microscopic and a macroscopic plane and we can avoid them only after we have learned to recognize them.

It is irrelevant here to discuss the normal histology of a nerve. It is nevertheless essential that we should be fully cognizant of it, also the arrangement of the blood supply to a nerve, and the histopathological findings seen in this structure either as a result of section or compression. It is important to recall the statement by Erb[8] in 1876, namely, "Even a slight mechanical action may so change the molecular constitution of the motor nerves as to abolish their power of conduction." Experience teaches that motor nerves offer less resistance to such lesions than the sensory ones do. The fundamental work of Lewis, Pickering and Rothschild[9] which was later verified and elaborated on by Denny Brown[10] express the opinion that pressure influences conduction only by means of local ischemia. What is ischemia? It is a local and temporary deficiency of blood, chiefly due to the contraction of a blood vessel. It is not the absence of circulating blood.

Let me explain: There are no major arteries specifically devoted to the supply of nerves,[11] each nerve large or small receives nutrient twigs from adjacent arteries, such nutrient vessels are short, and variable in size and many may be seen even with the naked eye, or particularly so under the dissecting microscope. On reaching a nerve these small local nutrient arteries divide into ascending and descending branches, which course in the epineurium. Sometimes such vessels pass deeply into the nerve and run between the fasciculi. From these primary divisions of the nutrient arteries secondary branches

are given off penetrating more deeply and dividing further. The vessels concerned are arranged predominately in a longitudinal fashion in the perineural or inter fascicular connective tissue. The infra fascicular plexus is common to all the nutrient arteries and is continuous throughout the length of the nerve. It is fed or reinforced at various levels by the nutrient arteries in such a way that no one nutrient artery may be considered as dominating any portion of this plexus. This arterial supply to a nerve can be definitely seen under the dissecting microscope, and can be destroyed; if a nerve is roughly and carelessly stripped from its bed these delicate channels are likely to be torn, interrupting thereby the superficial system on the surface of the nerve, a disturbance which in time of itself is liable to embarrass the intraneural circulation. The intraneural vascular bed of a nerve, therefore, is a continuous longitudinal system. The application of fundamental neurosurgical principles under the dissecting microscope and realization of the nature of the blood supply to a nerve is one of the explanations for the recovery in cases surgically treated with Bell's palsy. There undoubtedly has been some kind of an assumption that the blood supply of nerves was of no great moment; peripheral nerves have been treated surgically with a total lack of respect for the arteries or the veins associated with them, similar treatment has not been accorded to the gut or the central nervous system. The pattern of vessels in nerves, however, is peculiar and is so disposed as to allow extensive mobilization without essential devascularization, if handled properly.

Until it is definitely established that peripheral nerves[12] are normally dependent upon the maintenance of their normal blood supply and that the interruption of this may result in pronounced physiological effects it is scarcely justifiable to assert that vascular disorders of nerves may alone be responsible for the various clinical phenomenona, flaccid paralysis or disorders of sensibility or both which characterize the so called ischemic neuropathies. However, when the compression involves a nerve which lies in direct relationship with a bony surface the ischemia may then be complete, since in that case the effects of the compression occurs simultaneously and uniformly over the whole area occupied by the nerve. As a result of the physiological effects of ischemia, partial or complete, of a localized portion of a nerve trunk as at the stylomastoid foramen there results a widespread disturbance of motor activity embracing the whole area of distribution of the nerve above and distal to the

ischemic zone. It is an accepted fact that ischemia involves the rapidly conducting fibres first. This is a possible explantion of pain in Bell's palsy in which a herpetic eruption is not observed, and I might add that pain contrary to what has been written is a very common symptom in this type of Bell's palsy and of the utmost significance. It is explained on the following basis: Impulses traveling along the slowly conducting fibres are released from the dampening effect of those passing in the rapidly conducting fibres. Severe pain results, so that stimuli which would not normally result in pain might become effective when the propagation of the impulse along the rapidly conducting fibres is abolished by ischemia.

It seems desirable to stress, however, that peripheral nerves do not have a privileged autonomy and are independent of their blood supply.[13] The importance of the blood supply is expressed in a flexibility of the vascular arrangement which permits the maintenance of a normal circulation despite the loss of much of the usual source of supply. The results of clinically and experimentally produced ischemia of a nerve segment disclose the functional unit as nerve fibre, plus its blood supply, and so reveal the necessity of the existing vascular design, able to adapt itself to varying conditions.

The intraneural plexus[11] forms a system which is normally reinforced by the local nutrient arteries, but which can function in a given length of nerve even when the local branches are ligated. Obliteration of epineural or intraneural vessels produces a degeneration of varying degree depending on the extent of vascular deprivation. The complete and entire system of vessels upon and more particularly within the nerve is functionally most important.

The following preliminary investigation was prompted chiefly by the fact that during the decompression operation for Bell's palsy, the nerve bundles in the affected facial nerve are seen to slowly buldge forth when the pressure is released with the slitting of the epineural and perineural sheath covering.[17, 18] This observation led us to believe that the nerve fibres were under pressure and could not expand, due to the constricting perineural sheath. Paralysis produced in this way was speedily relieved with recovery following slitting of this sheath down to and including the stylomastoid foramen point of exit of the facial nerve.

The cause of this edema is unknown, but following its production a dysregulation of local circulation occurs presumably with

death of the axonal elements. The axones can once more re-establish their continuity, providing the cells of the sheaths of Schwann maintain patent syncytial channels. If there is delay in this repair process or continued obstruction to axonal regrowth, the endoneural elements could proliferate with impenetrable scar formations and would produce a complete obstruction.

It was felt that a complete study of nerve circulation was beyond the scope of this investigation and was reserved for a future publication, but an attempt was made to reproduce changes that appear related to this condition.

METHODS

Previous investigators have studied peripheral nerves lying in soft tissue, however, here we are dealing with a cranial nerve, which in a great part of its total length is completely encased in bone. The nerve with its epineural arterial, venous and lymph supply occupies about two-thirds of the bony canal, but at the stylomastoid foramen the bony diameter is much smaller. Here the contents of the canal are tightly bound down by a fibrous connective tissue sheath[19] adherent to the epineurium so that any swelling from within would in all probability affect the venous return, with blocking of the lymph drainage, an irreversible change would occur, unless relieved.

The facial nerve of the rat was picked for easy accessibility and because it resembles the human in its course, branches, and blood supply. Several rats were injected with latex directly into the heart and the small stylomastoid artery could be seen to enter the stylomastoid foramen lateral to the nerve trunk. A small inconstant branch from the occipital artery entered the foramen posteriorly in one animal. The venous drainage was not as easily studied. Experiments were carried out on the right side of the face and the left nerve was used as a control. Experimental follow-up study was carried out by observing the produced facial paralysis and by the faradic current applied to the skin at fifteen volts or on the exposed nerve at ten volts. It was found exposure of the nerve gave much more accurate data. Color films were taken to record these reactions as definite proof of the reactions obtained. Anaesthesia consisted of intraperitoneal evapol reinforced by ether, if desired. The work was carried out under a dissecting microscope (20x) when the epineural vessels were being studied. Sections were placed in ten percent neutral formalin rather than normal saline to prevent distortion and sectioned

in paraffin using hematoxylin and eosin, and osmic acid stain. Special stain was used only when deemed necessary. Animals were killed from periods of 4, 24, to 48 hours, and from 2, 4, to 6 days up to 4 months.

Due to the possible occurrence of edema[20] as a constant factor in the production of paralysis two methods of study were used to artificially produce a dysregulation in the circulation of the nerve.

1. Cold was applied to the shaven face of the animal over the maxillary and mandibular branches of the nerve approximate to the stylomastoid foramen utilizing ethyl chloride, a Dundas Grant tube or a cube of ice, the latter was found to be constant and simple in application.

2. A suture was tied around the main trunk following the pattern of Cajal, but here the results varied from previous investigators, since the proximal edema extended up to include the stylomastoid foramen completely blocking its exit. If this ligature is left in place it produces the degree of damage comparable to the experiments of Denny Brown and others who used a clip or pressure cuff with accompanying loss of function. Further experimentation is being directed toward controlling the blood supply after the methods of Torraca, Durward and others.

RESULTS

By injecting trypan blue .2 cc intravenously, twenty-four hours prior to the experiment and at operation .3 cc intraperitoneally, a generalized blue color was obtained in the blood vascular system. A cube of ice was placed on the right shaven face of a rat for twenty minutes, twenty-four hours later under the dissecting microscope the blood vascular supply of the facial nerve was studied. On the side where ice was placed there was a definitely bluer tinge than on the normal side, suggesting an increase in tissue fluid stained with trypan blue. The vessels coursing through the epineurium and down into the perineurium could be followed and it was obvious that a great increase in vascularization had occurred in Fig. 2*B* as compared with the normal Fig. 2*A*.

The application of ice to the face in other animals showed a temporary loss in conduction in response to faradism lasting a few minutes followed by a continuing normal response from then on. However, microscopic sections up to twenty-four hours showed no

Fig. 2.—*A.* A drawing to show the distribution of vessels on the normal facial nerve of a rat, maxillary division, as viewed through the dissecting microscope (20x) following intraperitoneal injection of trypan blue.

B. A drawing to show that 24 hours after the application of ice for 20 minutes to the skin overlying the nerve, there has occurred a great increase in vascularity with staining of the sheath from subperineural fluid collection.

change, but from there onward a collection of fluid was seen to have pushed the funiculus over to one side and away from the perineurium as seen in Fig. 4*B*—twenty-four hours after the experiment. The control side did not show this change. Further experiments at forty-eight hours and four days postoperative showed some separation of the endoneural segments and a small space only beneath the perineurium. The finding of this collection of fluid in the nerves examined must be considered with certain reserve, since the circulation to nerves is not uniformly established. There was no damage noted in the funiculus, nor was any expected, since this was not an irreversible change, but a slight degree of axonal swelling was present.

In the second series of experiments a suture applied to the nerve just below the stylomastoid foramin produced obvious facial paralysis within a matter of hours or days, depending on the tightness of the suture. Paralysis of the facial muscles was accompanied by a loss in faradism. The most interesting finding was the ever present gross edema above the suture extending to the stylomastoid foramen as shown in Fig. 3.

The suture caused a constriction and produced death of the axons proximal to the obstruction and distal to it. Osmic acid stain revealed myelin breaking down to form droplets in the proximal segment

Fig. 4*C*—with a more pronounced breakdown in the peripheral segment as shown in Fig. 4*D*. The breakdown in the proximal segment is more obvious in the longitudinal section Fig. 4*E*.

Fig. 4*F* illustrates myelin still in the process of absorption 30 days later, in this case the suture was removed two weeks after the initial operation. In another experiment the suture was removed after four weeks and faradism was checked weekly thereafter. The maxillary division showed return of function three months later, but little or no reaction present in the temporal or mandibular portions using fifteen volts. The post auricular branch was intact, since the suture did not include this branch.

DISCUSSION

Any trauma to a nerve results in a local interference with normal blood supply, however, as seen in Fig. 4*A* a constant large arteriole maintains adequate intrafasicular nourishment despite external damage to vessels. The intrafasicular venous system is of capillary dimension so that pressure from without either direct by stricture or indirectly by edema production can cut off the venous return with subsequent tissue damage. Since the nutrition of all cells depends on an even balance between hydrostatic (mechanical) and osmotic pressure in the regulation of tissue fluid, any change progressing too far in one direction caused by external compressive states may result in an irreversible state.

In these experiments intrafascicular edema was produced by the application of cold. If one adds a constricting band externally as exists at the stylomastoid foramen the interfascicular vessels are blocked leaving only the central vessels for nourishment. Theoretically the central arteriole can withstand this increase in pressure, but the accompanying precapillary venule and associated capillaries would be compressed with more tissue fluid collecting. This allows the blood to come in, but adequate drainage cannot be provided. In all our sections the arteriole was present and functioning, but in the cases where the ligature was applied its diameter was reduced distal to the ligature, and slightly engorged proximal to the suture. This situation is not observed so dramatically in a peripheral nerve since collateral circulation is slowly established.

With the use of a ligature the speed of breakdown and repair is greatly slowed; a relative ischemia is present in comparison to com-

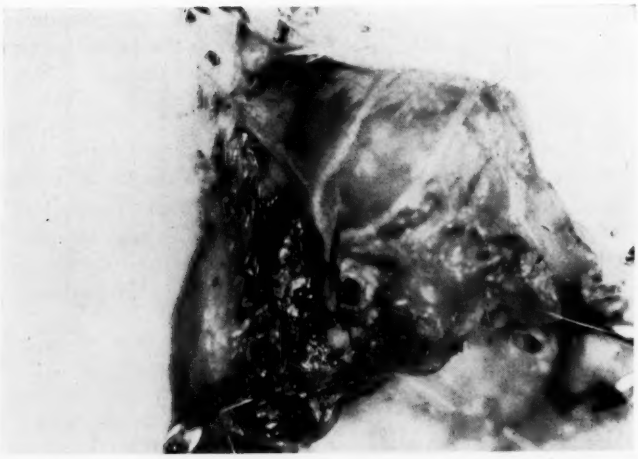

Fig. 3.—A photograph to show the edematous portion of the facial nerve proximal to a constricting suture and extending to the stylomastoid foramen.

plete division of a nerve. It is felt that a similar situation occurs in Bell's palsy so that early re-establishment of normal circulation by decompression at the stylomastoid foramen will hasten the recovery Fig. 5.

Denny Brown[10] believes that the damaging effect of varying pressures may be related to the occlusion of the blood vessel of a nerve. His experiments of compression by a clip along with ours is a facsimile of what occurs acutally in the region of the stylomastoid foramen of the facial nerve with Bell's palsy. The structural alteration at the site of compression is looked upon as passive distortion consequent on the vascular narrowing with congestion above and below. This fact has been very reassuring to me because it is a belief that I have held for many years. It is likely that any form of pressure applied to a nerve will by angulation and unequal stress cause physical injury, besides the ischemia produced in pure form by experimental compression, so that Wallerian degeneration of the fibres will occur. The defect of block in conduction produced by a compression is selective, that is, whether it is of a minor or a very severe

degree in type. This variability is closely related to variable gradients of pressure in the tissues where anatomic structures provide compartments of different rigidity.

ELECTRICAL REACTIONS

Muscle atrophy and the resulting fibrotic changes are soon apparent in the thin musculature of the face where there is no definite muscle fascia surrounding the individual muscles. These are inserted into subcutaneous tissue or skin without the intervention of any subcutaneous fascia. When denervation has persisted for any length of time the fibrotic sheath which replaces the individual muscle cannot be made to contract even if continuity of the nerve could be restored. The importance of physical treatment of the facial muscles in every patient with seventh nerve palsy cannot be over estimated. The best surgical technique which will restore conduction of the nerve impulse is useless if the facial muscles have been allowed to stretch and atrophy before the operation or while nerve regeneration is taking place.

Faradism. A normal muscle responds to percutaneous faradic stimulation at low intensities.[14] If the nerve degenerates its excitability decreases and is ultimately lost. It is taken for granted that we realize that faradism is a direct stimulation of short duration to nerves. The muscle contracts because it is receiving impulses via the intramuscular nerve twigs. Galvanism is a direct stimulation to muscle tissue. The galvanic response is much more easily elicited. Failure to elicit a galvanic response in muscle in the absence of edema indicates serious atrophic change in a muscle. Fibrous tissue seem to wage a war of attrition upon muscle fibres.

Electromyography. From its history it is obvious that electromyography[15] as it is known today is a direct application of electrophysiology to clinical medicine. The action current of contracting muscles can be recorded. The electrocardiogram is a well known example. By electromyography it is possible to record the action current in a voluntary muscle. After section or severe compression of a nerve the muscle fibres supplied by it naturally lose their power of contracting on volition as well as the collective electrical disturbances associated with this. It does not, however, spell the end of activity of the individual muscle fibres composing the motor units. After a lapse of approximately three weeks these fibres may begin to enjoy

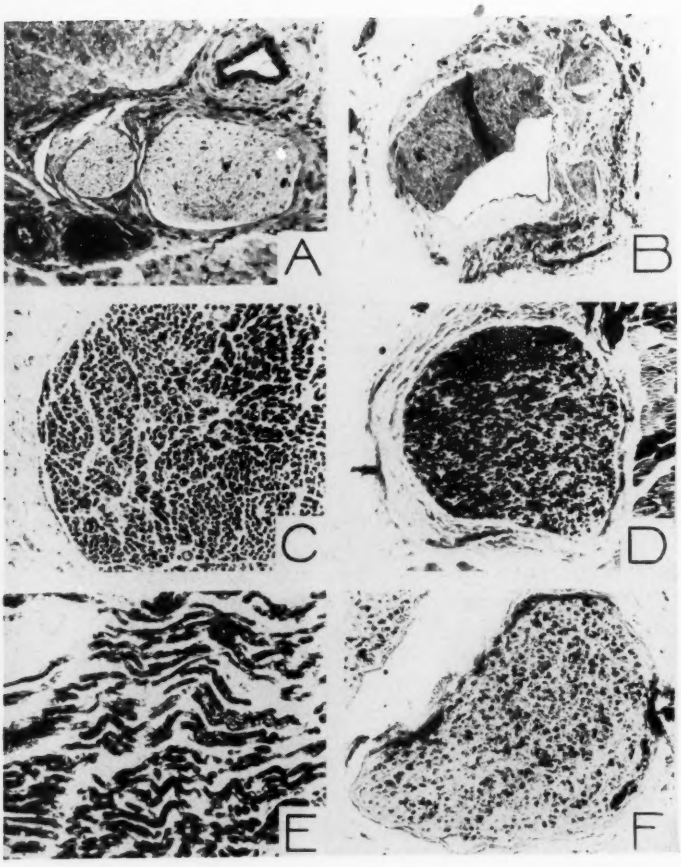
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Fig. 4.—*A.* A low power photomicrograph of a section to show the maxillary division of the facial nerve of a normal rat with its accompanying artery, vein and lymph duct; in this division the parotid duct is also present. Note the constant more or less central intrafascicular arteriole in the endoneurium. A capillary plexus is visible under oil immersion (H & E).

B. A low power photomicrograph of a section of the facial nerve of a rat to show a collection of fluid beneath the perineural sheath producing distortion of the nerve bundle 24 hours after the application of ice for 10 minutes to the skin overlying the nerve. Under oil immersion the axis cylinders appear full (H & E).

C. A medium power photomicrograph of a section of the facial nerve of a rat proximatel to a constricting suture, 9 days post-operative. There is patchy retrograde degeneration of axonal and myelin elements with myelin sheath swelling. There is a moderate amount of edema fluid in the endoneural spaces. Regeneration is barely suggested. (Osmic Avid)

D. A medium power photomicrograph of a section of the facial nerve of a rat distal to a constricting ligature, 9 days postoperative. Here there is frank myelin degeneration with the exception of one area which appears to have escaped compression. Ischemia is present. This stage resembles the 2 day postoperative nerve following section and illustrates a slower process of similar magnitude. (Osmic Acid)

E. A high power photomicrograph showing the proximal portion of the nerve in longitudinal section 10 days after the application of a ligature. This section is closer to the suture area and shows myelin degeneration with droplet formation. The axis cylinders show fragmentation. There is separation of the fibres by edema fluid collection. Again the distortion is similar to that seen 2 days after section of a nerve. (Osmic acid)

F. A high power photomicrograph of the facial nerve in cross section just distal to the ligature 30 days after operation. In this case the ligature was removed 14 days after the original operation. Here old myelin is in evidence. The nerve fibres are large and appear to have coalesced in some cases with endoneural proliferation. Regeneration has started and a few axis cylinders are getting a new coat of myelin. Here degeneration and regeneration is progressing at the same time unlike a section of the nerve. There was no reaction to faradic stimulation. (Osmic Acid)

an autonomous life of which one manifestation is a rhythmic activity termed, "fibrillation." As a result of muscle study, it is revealed, that surgical repair, if necessary, should be undertaken as soon as possible. In our experience the interpretation of electromyography results is strictly limited in Bell's palsy.

Weddell[16] states: that the most difficult muscles to relax are those of the face, tongue and abdominal musculature. The presence of fibrillation is considered of great diagnostic and prognostic significance in peripheral nerve injury, however, there are other symptoms in differentiating quantitatively between complete and partial denervation and qualitatively between paralysis due to transient nerve block and that due to axon degeneration. At the moment electromyography may well be possible in the future to standardize recording technique in terminology to such an extent as to make this practicable, but the time has not yet come, furthermore, the clinician irrespective of the findings by this method can confirm or reject a tentative diagnosis either on clinical grounds or as a result of an exploration.

Erb was the first to point out the importance of electrical reactions and stated that, in certain circumstances a brisk galvanic and faradic response of individual muscles may still be obtained where a large number of axons have been interrupted. In a case of inexcitability of a nerve below the level of the lesion it seems not improbable that the retention of the faradic response is related to the presence of the few undegenerated nerve fibres.

In some cases of Bell's palsy it is difficult to obtain an accurate result by the faradic test, this is due to an unduly high skin resistance. The faradic response may not be obtained in this case and yet when the nerve is exposed and an electrode applied directly to the nerve conductivity can be demonstrated. For this reason the nerve should be exposed and stimulated in all questionable cases; another finding that lends itself to this important prerequisite is the very fact that pain is a factor in faradic stimulation and as I have previously stated on many an occasion, it is why in order to be of a degree of finality the test should be carried out under an anaesthetic. Weddell[16] states, "It is not possible by electromyography to distinguish between a total axonal interruption and a complete ischemic block for some days after the injury." These observations Weddell has made, have not included many cases of Bell's palsy where their findings for a great ma-

Fig. 5.—*A.* Photograph of facial nerve (human) in Bell's palsy—permission of T. Cawthorne. *B.* Explanatory drawing of Figure 5*A*.

jority of these cases were quite unspecific. He goes on further to state, that ischemic block may persist in unlimited periods and in such a case recovery might be expected to follow decompression, unless a persisting infection is present. There is experimental and clinical evidence that atrophy of muscles can be retarded by activity induced by adequate electrotherapy. The contraction of the muscle is essential, therefore, faradic stimulation of denervated muscle is useless and a waste of time. Denervated muscle responds to galvanism and this is, therefore, one method of treatment.

Some claim that vasospastic states can produce changes in nerves. This applies much more to a sensory nerve, such as the fifth nerve than it does to a motor nerve, such as the facial nerve. For example, we have seen in patients with severe fifth nerve neuralgia the administration of a vasodilator drug, which in some cases has helped. Also the possibility of involvement of the eighth cranial nerve and its end organ in the typical condition of hydrops of the internal ear, but this is a vasospasm[21] within the fifth or the eighth nerve at the root of a sensory disturbance. The argument which has been presented in the latter has been based on examination and experience with sensory nerves. I do not feel that this applies to the pure motor facial nerve. Realization of a primary vascular lesion producing a nerve paralysis opens up a vista of explanations of common and frequent nerve lesions. A Bell's palsy may well be due to a dysregulation of the artery to the facial nerve, or a localized thrombophlebitis producing a paralytic lesion of the nerve. Recovery ensuing with opening of the artery or the establishment of a collateral circulation in the case of a vein. The satisfactory recovery of a facial paralysis after decompression of its bony canal may be explained on the basis of permitting an early and effective collateral circulation to combat the effect of a previously occluded vessel. A parallel exists in the fact that a cortico-thrombophlebitis will produce paralysis of a limb and recovery follows the establishment of a collateral circulation.

Local constriction of a nerve makes it impossible for those parts of the nerve fibres distal to the affected level to regain proper caliber and myelinization. Excessive contraction produces pressure block of conduction through the strangled region of the stylomastoid foramen. Expressed simply: The nerve swells in its canal, the nerve fibres cannot convey impulses owing to the spread pressure and paralysis results.

SUMMARY

The damaging effect of pressure on a nerve is due to occlusion of the blood vessel and here I am in agreement with Collier.[22] This dysregulation of the circulation as I prefer to call it effects primarily the myelin sheath, has at first little, if any, influence on the Schwann cells and axis cylinders. Here is the temporary block to conduction, but continuous pressure produces other changes. Vascular stasis occurs in neighboring segments leading to edema above and below the site of compression as we have shown. Not only is the process of repair in the compressed area impeded but a complete degenerative lesion involving the axis cylinders and Schwann cells is finally produced. It is thus seen how reversible ischemia block which may be present for lengthy periods in nerves of the limbs without leading to degeneration, will by persisting in the rigid facial canal produce increased vascular stasis and edema leading to complete degeneration. Where we have pain accompanying complete facial paralysis, is indicative in itself, of a severe degenerative lesion and to wait for unequivocal evidence of degeneration, even with direct exposure of the nerve to elicit the faradic reaction which cannot be conclusive for fourteen days would condemn some patients to the limited imperfect recovery which accompanies regeneration.

What is the fundamental neurosurgical principle involved in crushing injuries involving nerves and bones anywhere in the human body, but that of relieving the pressure on the involved nerve and so mobilizing the nerve to allow it to regain its functional condition as soon as possible. If that is the principle involved in soft tissues what must it be of a motor nerve encased in a bony canal? It has been revealing to me over a period of twenty years in the examination of this nerve under a dissecting microscope, the extreme degree of edematous swelling that immediately shows itself with marked swelling of the nerve bundles, becoming manifest after the careful removal of the upper two-thirds of the bony canal without damage to the blood supply as far as it is surgically possible, and incising the epineural connective sheath along the side of the nerve instead of on its upper aspect in order to prevent damage to the longitudinal arterial anastomosis. This appearance has been observed anywhere from three weeks up to a period of twelve weeks, and particularly is it so above the area of the actual foramen itself with less manifestation of this macroscopic change as the nerve extends out-

wards and divides at the pes anserinus. The rapidity of recovery following this procedure is only too self-evident.

I make no apologies for suggesting the early decompression of the nerve in Bell's palsy in which by actual examination as described we have revealed the absence of the faradic response. The appearance of the nerve in cases anywhere from six months to a year following this change in which all that has remained has been a fibrous strand more than anything else vindicates the rationale of this procedure.

Merwarth[23] states that recurrences of facial paralysis are fairly common, and particularly is this so in Bell's palsy. It is, therefore, interesting to recall the fact that in all my cases that have been subjected to surgical intervention no recurrence of facial paralysis has resulted on the operated side; similarly that finding is true in the cases of Cawthorne of England.[24] This means, that compression, in addition to the dysregulation of the circulation, is the cause or the factor in the production of this lesion in the seventh nerve.

We are dealing with a cranial nerve, a motor nerve, which has to do with muscular power and emotional expression, with facial muscles lacking a fascial covering and prone to rapid muscular atrophy. If the triad of symptoms I have just enunciated is present you cannot decompress the facial nerve too early.

1. Bell's palsy as we understand it in the human may be considered as an edematous neuropathy, resulting from a dysregulation of the circulation.

2. The primary cause is as yet unknown, the results known.

3. That mechanical factors are much more important in the production of this lesion than previously appreciated.

4. A method of experimentation is presented to simulate the changes believed to take place in Bell's palsy as we understand it in the human wherein a dysregulation of circulation at the stylomastoid foramen can produce edema with damage of function.

5. The application of these findings in the rat are presented with reservation, since similar human experimental studies are not feasible.

6. That other experimental work in dealing with peripheral nerves has not encountered similar compartments of rigidity peculiar to the facial nerve.

7. Where the faradic response is inconclusive by testing through the skin, it is suggested that direct exposure of the nerve should be carried out to establish with certainty the presence or absence of this reaction.

8. A Bell's palsy presenting complete facial paralysis, loss of the faradic response, accompanied by pain, is indicative of a severe degenerative lesion and warrants immediate surgical interference.

CONCLUSION

Scott Stevenson[25] in his "History of Otolaryngology" writes: "Although we live in the days of antibiotics and isotopes, of vitamins and chemotherapy, of electronics and endocrinology, the Golden Age of otolaryngology is yet to come—when the tonsils cease from troubling and the sinuses are at rest; when doctors send their patients and their Bell's palsy patients to the otolaryngologist too early rather than too late, when diagnosis can be considered as precisely accurate, when all infection can be effectively controlled, when cancer and tuberculosis are conquered at last and when deafness can be cured."

The writings of the ancient authors clearly show a knowledge of certain broad clinical facts respecting diseases of the ear, and the complications which arise there from. It is not by the narration of examples alone, that medicine or surgery becomes an aid in the study of a new clinical entity, it is essential in relation to our subject to catch the character and spirit of the age from which the example is drawn, then in the light of history, the present, and future, may be interpreted and guessed at. Although the whole meaning lies far beyond our ken, yet in that complex manuscript, here and there, still dimly legible, an intelligent precept available in practice may be gathered.

My sincere thanks are due to Professor Arthur W. Ham of the Department of Anatomy of the University of Toronto in whose laboratory the experimental work was carried out, Professor Vera Bensley was kind enough to corroborate the microscopic findings, to J. K. B. Smith our experimental surgeon, and research fellow in the Department of Otolaryngology, and to P. E. Ireland, Professor of Otolaryngology, of the University of Toronto, I would like to express my gratitude for their contributions.

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Abstracts of Current Articles

EAR

Reactive Post-contusional Deaf-mutism. Its Diagnosis and Treatment.

Perelman, L. B.: The State Publishers, Narkomzdrava for Medical Literature, U. S. S. R., Moscow, 1943.

The author reports his experiences, as a psychiatrist, with approximately 300 cases of deafness and deaf-mutism, all of a hysterical nature, appearing in soldiers exposed to detonation of shells during the war.

All of his cases are functional in character, and represent hysteria, although some of the patients had associated physical injuries, and disabilities on an organic basis. The study does not include blast injury to the auditory apparatus. In differentiating the hysterical deafness following exposure to detonation from true blast injury to the ear with deafness, some very interesting points are made: true loss of consciousness occurred in over 90 percent of the blast injury cases, but true loss of consciousness was rarely found in the hysterical deafness cases. These latter patients observed vividly all the attending circumstances of the explosion; indeed, it became a fixation with them. In the hysterical patients there was always total bilateral deafness, whereas this only occurred in 28 percent of organic injury cases.

The author states that in all cases there will be a certain amount of organic damage to the auditory apparatus, but whereas usually there is some improvement in the non-hysterical subject, the hysterical person becomes "fixed" on the ear condition, and the "organic" changes over to "functional." The emotional trauma of combat serves as a powerful factor in this change.

In contrast to patients suffering from actual loss of hearing, the hysterically deafened person does not make any effort to understand speech directed toward him; there is lacking, so to speak, the aspect or attitude of the deaf, the yearning to read lips.

In 48 percent of the cases there was deafness and mutism; in 12 percent of the cases mutism alone; in 40 percent deafness alone. The mutism bore the characteristics of the well-known hysterical aphonia. Frequently other hysterical manifestations were present: paralyses, anaesthesias etc.

Experience showed that the hysterical deaf person can actually hear and perceive physical sound and also parts of speech, particularly in those cases where the final portion of the speech is not directed straight at the patient, and where his attention was diverted from this speech. This was made the basis of an examination by the author, which he called "combined testing," in which the patient is shown a series of printed or written questions, which are at the same time read aloud to him; as the tests progress, the printed questions each commence just as spoken ones, but the latter part of the sentence is different in the printed and in the spoken form. The hysterical patient is "tricked" into answering the spoken question, thus revealing his hearing acuity for the spoken sentence. Later the printed form becomes quite far removed from the spoken form, indeed nonsensical, yet the hysterical person replies to the spoken question.

A second interesting diagnostic test consists in the setting up of a "conditional reflex" experiment, after the manner of Pavlov. A small electric shock is presented to the patient's hand, simultaneously with an audible tone. The process is repeated until a "conditioned reflex" is established. Then, by presenting the tone alone, the hysterically deaf person will quickly move his hand, just as though he had received the electric shock. Naturally, such a reflex can not be worked out for a truly deaf patient.

Considerable time is given to abstract discussion of psychiatric principles and, to an American, this Russian monograph seems unnecessarily wordy. However, it gives an interesting insight into hysterical deafness.

Treatment is purely psychiatric, its aim being to break down the "ear-fixation" and to restore the patient's confidence in himself.

HARKINS.

NOSE

Treatment of Antral Cancer by Combined Surgery and Radium Therapy.

Collins, V. P., and Pool, J. L.: Radiology 55:41 (July) 1950.

The authors describe in detail treatment of antral cancer. A peroral resection of the antral floor with a limited antrotomy or a radical resection of the maxilla is done. Within ten to fourteen days an impression of the defect is taken in dental compound and a radium applicator is made therefrom in clear dental acrylic. Zones to be irradiated are mapped on this mold. These areas are then drilled

to accept the calculated sources of radium. Roentgenograms in three dimensions at two meters distance demonstrate the position of the radium sources after the radium applicator has been retained in position by a modified Kingsley splint. X-ray therapy may be necessary for cases with extensive involvement.

Diagrams illustrate the operative procedures. Photographs illustrate the roentgenogram localization of the radium source. Photographs of the acrylic mold and graphs of radium dosage for point sources are included.

JORSTAD.

Treatment of Carcinoma of the Nasopharynx by Irradiation.

Eberhard, T. P., and Leaming, R. H.: Radiology 55:46 (July) 1950.

The authors emphasize importance of visualizing the tumor of the nasopharynx and making careful radiographic examinations to depict early bone destruction. Biopsy should be obtained when possible. These factors along with general examination of patient are important considerations in planning treatment. The ideal goal of treatment is the administration of a minimum of 4,000 to 4,500 r to all parts of the tumor in four weeks. The primary area is usually treated through four fields and if necessary this may be supplemented by radium with a suitable nasopharyngeal applicator. Lateral fields are used in delivering x-radiation to the cervical lymph bearing areas. Details of treatment are discussed and illustrated by schematic drawings. End results of treatment are given.

JORSTAD.

MISCELLANEOUS

Surgical Management of Lymph Node Metastases.

Taylor, G.: Radiology 55:60 (July) 1950.

The author discusses the fallibility of depending upon the clinical estimate of lymph node metastases and illustrates this in a table demonstrating the size of cervical lymph nodes in relation to actual metastatic involvement secondary to carcinoma of the lip. The relationship of the characteristics of the primary carcinoma of the lip to the occurrence of metastases is tabulated and similar studies of carcinoma in other sites are discussed. Further tables show percentages of cases with positive nodes cured by neck dissection as related to the clinical evaluation of the nodes and the site of the primary lesion, the primary lesion having been cured.

JORSTAD.

Treatment of Cervical Lymph Node Metastases with Irradiation Alone.

Martin, C. L.: Radiology 55:62 (July) 1950.

The author discusses results of neck dissection for metastatic carcinoma reported by various observers. He evaluates radiologic technics used. He presents evidence to prove that metastatic cervical lymph nodes secondary to squamous cell cancer of the head, neck, and mouth can be eradicated by a combination of interstitial radium (low-intensity needles) therapy and external x-ray therapy applied simultaneously during a period of seven days. A statistical survey of a series of 146 cases diagnosed clinically and treated with this technic five or more years ago suggests that the salvage falls in the same range as that reported for block dissection.

JORSTAD.

Supervoltage Roentgen Therapy in Cancer of the Mouth and Throat.

Schulz, M. D.: *Radiology* 55:52 (July) 1950.

The author considers the use of supervoltage radiation in treating malignant lesions about the mouth and throat. The twelve years' experience with supervoltage radiation at the Massachusetts General Hospital and Collis P. Huntington Memorial Hospital has borne out the conclusion that, with few exceptions, it has not been spectacularly more effective than x-rays generated at 200 to 250 kv., all other things being equal. He discusses supervoltage therapy from the standpoint of (1) its effectiveness in ridding the patient of cancer and (2) the degree of harm to normal tissue required to accomplish this.

JORSTAD.

Books Received

II Diabete Mellito in Otorinolaringoiatria.

By Prof. F. Vigi, Prof. G. Lenti, and Prof. L. Cojazzi. Pp. 144. Arcispedale S. Anna - Ferrara.

The Antihistamines.

By Samuel M. Feinberg, M.D., Associate Professor of Medicine, Chief of Division of Allergy and Director of Allergy Research Laboratory, and Saul Malkiel, Ph.D., M.D., Assistant Professor of Medicine, Director of Research, Allergy Research Laboratory, and Alan R. Feinberg, M.D., Clinical Assistant in Medicine, Attending Physician in Allergy Clinic. Pp. 291, with 27 iilustrations and 2 tables. Year Book Publishers, Inc. Chicago, Illinois, 1950. (Price $4.00)

Atlas of Human Anatomy.

By Barry J. Anson, Ph.D., Professor of Anatomy, Northwestern University Medical School. Pp. xxi+518 with 500 Illustrations. Philadelphia, Pennsylvania, W. B. Saunders Company, 1950.

Tumors of the Head and Neck.

By Grant E. Ward, M.D., D.Sc., F. A. C. S. and James W. Hendrick, M.D., M.S. From the Departments of Surgery of the School of Medicine, University of Maryland, and the Johns Hopkins Hospital School of Medicine, and Oncology Clinic of the University Hospital and the Tumor Clinic of the Johns Hopkins Hospital. Pp. xv+832, with 637 figures and 46 tables. Baltimore, Maryland, The Williams & Wilkins Company, 1950. (Price $15.00)

Bronchoesophagology.

By Chevalier Jackson, M.D., Sc.D., LL.D., F. A. C. S., Honorary Professor of Bronchoesophagology and Laryngeal Surgery, Temple University, Philadelphia, and Chevalier L. Jackson, M.D., M.Sc., F. A. C. S., Professor of Bronchoesophagology and Laryngeal Surgery, Temple University, Philadelphia. Pp. 366, with 268 figures. Philadelphia and London, W. B. Saunders, 1950.

Your Deaf Child.

By Helmer R. Myklebust, Ed.D., Professor of Audiology, Northwestern University. Pp. xv+133 with 17 figures. Springfield, Illinois, Charles C. Thomas, 1950. (Price $2.50)

A Syllabus of Laboratory Examinations in Clinical Diagnosis.

Edited by Thomas Hale Ham, B.S., M.D., Assistant Professor of Medicine, Harvard Medical School: Associate Director, Thorndike Memorial Laboratory; Junior Visiting Physician, Boston City Hospital. Pp. xii+496. Cambridge, Massachussets, Harvard University Press, 1950. (Price $5.00)

Aphorisms From His Bedside Teachings and Writings.

By Sir William Osler. Pp. 148. New York, Henry Schuman, Inc., 1950. (Price $2.50)

The Physician Examines the Bible.

By C. Raimer Smith, B.S., M.D., D.N.B. Pp. vii+394. New York, New York, Philosophical Library, 1950. (Price $4.25)

Language for the Preschool Deaf Child.

By Grace Harris Lassman, Teacher of the Deaf; formerly Instructor of Speech, John Tracy Clinic, Los Angeles, California. Pp. xviii+263 with 27 illustration. New York, New York, Grune & Stratton, Inc., 1950. (Price $5.50)

Einführung in Die Hals-Nasen-Ohren-Heilkunde.

von Dr. Med. Paul Falk, Ordentlicher Professor Der Oto-Rhino-Laryngologie und Direktor Der Hals-Nasen-Ohren-Klinik Der Universität Des Saarlandes in Homburg (Saar). Second Edition, with 150 illustrations some in color. Stuttgart, Georg Thieme Publisher, 1950.

Food Allergy.

By Herbert J. Rinkel, M.D., F.A.C.P., F.A.C.A., F.A.A.A., Formerly, Associate Instructor in Medicine, University of Oklahoma, School of Medicine, Theron G. Randolph, M.D., F.A.C.A., F.A.A.A., A.A.C.P., Instructor in Medicine, Northwestern University Medical School, and Michael Zeller, M.D., F.A.C.P., F.A.A.A., Clinical Instructor in Medicine, University of Illinois College of Medicine. Pp. xv+497 with 25 illustrations and 182 recipes. Springfield, Illinois, Charles C. Thomas, 1951.

Notices

NOTICE

The American Board of Otolaryngology will conduct the following examinations in 1951:

May 1-4, in Richmond, Virginia, at the Hotel John Marshall.

October 9-12, in Chicago, Illinois, at the Palmer House.

DEAN M. LIERLE, M.D., *Secretary*
University Hospital,
Iowa City, Iowa

NOTICE

The Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology will hold its Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology in Havana, Cuba, in 1952.

For information address: Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pa.

UNIVERSITY OF TORONTO

The University of Toronto, Faculty of Medicine, announces a combined Refresher Course in Ophthalmology and Otolaryngology during the week of January 29th to February 3rd, 1951. Dr. Phillips S. Thygeson, University of California School of Medicine and Dr. Charles E. Iliff, Johns Hopkins University will be guest speakers in Ophthalmology. Dr. LeRoy Schall, Harvard University and Dr. G. B. Tremble, McGill University will be guest speakers in Otolaryngology. There will be surgical and medical clinics in these two subjects in addition to lectures by members of the Faculty.

The course will be given for a minimum of 10 students and a maximum of 25 students. Application may be made to the Dean of the Faculty of Medicine not later than November 30th, 1950.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION THE AMERICAN MEDICAL ASSOCIATION

(List Corrected to December 1, 1950)

Aurex Model F
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Beltone Mono-Pac Model M

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Cleartone Regency Model

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Electroear Model C

Gem Model V-35
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Maico UE Atomeer
Maico Quiet Ear Models G & H
Mears Aurophone Model 200
1947-Mears Aurophone Model 98
Micronic Model 101 (Magnetic Receiver)
Micronic Model 303 (See Silver Micronic)
Microtone T-3 Audiomatic
Microtone T-4 Audiomatic
Microtone T-5 Audiomatic
Microtone Classic Model T9
Microtone Classic Model 45

National Cub Model (C)
National Standard Model (T)
National Star Model (S)
National Ultrathin Model 504
National Vanity Model 506

Otarion, Model E-1
Otarion, Model E-1S
Otarion, Model E-2
Otarion, Model E-4
Otarion, Models F-1 & F-2

Paravox Models VH & VL (Standard)
Paravox Model XT ('Xtra-Thin)
Paravox Model XTS ('Xtra-Thin)
Paravox Model Y (YM, YC and YC-7 (Veri-small)

Radioear Permo-Magnetic Multipower
Radioear Permo-Magnetic Uniphone
Radioear All-magnetic Model 55
Radioear Model 62 Starlet

Silver Micronic (Magnetic and Crystal) Models 202M & 202C (See Micronic)
Silvertone Model 103BM
Silvertone Model M-35
Silvertone Model P-15
Solo-Pak Model 99
Sonotone Model 600
Sonotone Model 700
Sonotone Model 900
Sonotone Models 910 & 920
Sonotone Model 925
Super-Fonic Hearing Aid

Televox Model E
Telex Model 22
Telex Model 97
Telex Model 99
Telex Model 200
Telex Model 1700
Tonemaster Model Royal
Trimm Vacuum Tube Model 300

Unex Model A
Unex Midget Model 95
Unex Midget Model 110

Vacolite Model J

Western Electric Model 63
Western Electric Model 64
Western Electric Models 65 & 66

Zenith Model 75
Zenith Miniature 75

(All of the accepted hearing devices employ vacuum tubes.)
Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

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Precision Table Hearing Aid
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President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
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American Broncho-Esophagological Association

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Meeting: Greenbrier, White Sulphur Springs, W. Virginia, May 7 and 8, 1951.

American Laryngological Association

President: Dr. Gordon B. New, Mayo Clinic, Rochester.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Greenbrier, White Sulphur Springs, W. Virginia, May 9 and 10, 1951.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Greenbrier, White Sulphur Springs, W. Virginia, May, 1951.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.

American Otological Society

President: Dr. Kenneth M. Day, 121 University Place, Pittsburgh, Pa.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago, Ill.
Meeting: Greenbrier, White Sulphur Springs, W. Virginia, May, 1951.

II Pan-American Congress of Otorhinolaryngology and Broncho-Esophagology

President: Dr. Justo Alonso.
Secretary: Dr. Pedro Regules, San José 1092, Montevideo, Uruguay.

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